A Dissertation on

NON-SMOKING TOBACCO ABUSE PATTERNS AMONG PATIENTS WITH ESTABLISHED ORAL CANCER

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BONAFIDE CERTIFICATE

Certified that this dissertation is the bona-fide work of D**r. R. GOPIKRISHNAN** on "**NON-SMOKING TOBACCO ABUSE PATTERNS AMONG PATIENTS WITH ESTABLISHED ORAL CANCER"** during his M.S. (General Surgery) course from May 2011 to April 2014 at the Madras Medical College and Government General Hospital, Chennai.

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DECLARATION

I Dr. R.GOPIKRISHNAN declare that this dissertation titled "NON-SMOKING TOBACCO ABUSE PATTERNS AMONG PATIENTS WITH ESTABLISHED ORAL CANCER" represents a genuine work of mine. The contributions of any supervisors to this research are consistent with normal supervisory practice and are acknowledged.

I also affirm that this bona-fide work or part of this work was not submitted by me or any others for award, degree or diploma to any other university board in India or abroad. This work is submitted to The Tamilnadu Dr.M.G.R. Medical University , Chennai in part fulfilment of the rules and regulations for the award of Master of Surgery degree Branch I (General Surgery)

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ABSTRACT

BACKGROUND:

In the Indian scenario, a large percentage of tobacco abuse is in the form of Smokeless tobacco (ST) and the usage pattern has undergone a gradual change over the years with chewers becoming lesscommon.Hence, there is a need to reassess the epidemiology of oral cancer with respect to these recent tobacco product usage.

AIMS:

This study aims to find the usage pattern of non-smoking tobacco in patients with oral cancer in comparison with people without cancer.

MATERIALS & METHODS:

100 cases of established oral cancer patients with history of quid use were compared with 100 age matched controls with quid use but no cancer. Both were administered semi-structured questionnaires and data analysed with SPSS.

RESULTS:

The most common age group affected by cancer was 46-55 yrs. (35%), mean age of presentation was 51.5 years and 31% presented with cancer at age <45 yrs. The mean years of exposure for tobacco quids placed in mouth was 20.85 with significantly more quids use number, overnight usage pattern (69%) and placement for more than 1 hour/ usage (38%) among cases than controls. Other abuses like bidi (52%), cigarette (47%), alcohol (53%) and other types of smokeless tobacco (54%) were significantly more than in controls. Awareness of ill effects was lesser (53%) and abstinence was more in cases (54%) than in controls.

CONCLUSION

The study has found that a person who has oral cancer and primarily uses tobacco quids tends to use it more frequently, for longer periods of time than controls and is more likely to consume alcohol, cigarettes and bidis on a regular basis.The disfigurement and loss of function caused by oral cancer, its treatment, the number of life years lost due to the disease and the ever growing incidence of Oral squamous carcinoma in our country point to the fact that we need to emphasise on increasing public awareness, screening, early diagnosis and treatment.

Keywords: smokeless tobacco, abuse pattern, oral cancer, quid

INTRODUCTION

Oral cavity cancer is a huge health problem in India the recent increase in incidence being attributed to the wide availability of a variety of tobacco products in India. Tobacco abuse causes an enormous social burden, loss of productivity and myriad of health conditions. Tobacco use raises the chances for cancers of the mouth, lips, nasal cavity and sinuses, larynx, pharynx, lungs, oesophagus, stomach, pancreas, kidney, bladder, uterus, cervix, colon/rectum, ovary, and acute myeloid leukaemia. Tobacco is smoked, kept as 'quids' in the oral cavity and chewed alone or with various additives like lime, areca nut & betel leaves.

In the Indian scenario, a large percentage of tobacco abuse is in the form of Smokeless tobacco (ST). Ninety percent of oral cancers in India are attributable to tobacco making it the main culprit in the "Indian Oral Cancer" scenario. Although the causal role of smokeless tobacco has been well established in Oral squamous cell carcinoma, the myriad varieties of tobacco products & preparations with multiple unquantified ingredients in India pose a challenge in ascertaining the carcinogenic potential of these substances and establishing a dose response relationship. This is compounded by abusers resorting to concurrent use of tobacco through smoking, alcohol, poor oral hygiene which confounds such quantification.

Given below are some of the most common forms of smokeless tobacco from around the world.

TOBACCO	CONTENTS	REGION
Pan/paan/betel quid	Areca nut, betel leaf, slaked lime, catechu, condiments, with or without tobacco	India ,Southeast Asia, South America
'Khaini'	Tobacco, lime	Bihar
'Mishri'	Burned tobacco	Maharashtra
'Zarda'	Boiled tobacco	India, Arab nations
'Gadakhu'	Tobacco, molasses	India
'Mawa'	Tobacco, lime,areca	India
'Nass'	Tobacco, ash, cotton or sesame oil	Afghanistan, Pakistan, Iran,
'Naswar,niswar'	Tobacco, lime, indigo, cardamom, oil, menthol, etc	Asia,Iran, Afghanistan, Pakistan
'Shammah'	Tobacco, ash,lime	Saudi Arabia
'Toombak'	Tobacco,soda bicarb	Sudan

Apart from Bidis & cigarettes, numerous tobacco products like gutka, tobacco flakes are marketed in attractive sachets, ubiquitous and cheap, all of which contributes to their highly prevalent usage in India.

Consensus among medical workers and oncologists in standardising the definition of these different types of tobacco preparations is in process. For example, a workshop conducted in Kuala Lumpur recommended that "quid" be defined as "a substance, or mixture of substances, placed in the mouth or chewed and remaining in contact with the mucosa, usually containing one or both of the two basic

ingredients, tobacco and/or areca nut, in raw or any manufactured or processed form."(4) In addition to defining quid-related terminologies, the workshop participants set guidelines for reporting quid use among research subjects. They emphasised, the ingredients be outlined in detail so it would be possible to differentiate into three categories:

• "Quid with areca nut without any tobacco products involving chewing only the areca nut or areca nut quid wrapped in betel leaf ".

• "Quid with tobacco products but without areca nut, including chewing tobacco, chewing tobacco plus lime, mishri (burned tobacco applied to the teeth and gums), moist snuff, dry snuff, niswar (a different kind of tobacco snuff) and naas (a stronger form of niswar)".

• "Quid with both areca nut and tobacco products (paan with tobacco)".

Areca nut & tobacco are easily identifiable and hence were chosen to make product categorisation simple.

A study from India in concurrence with other smaller studies, concluded paan to be the most important causative factor of smokeless tobacco. The usage of smokeless tobacco has undergone a gradual change in India. Previously tobacco flakes were chewed and spat out. Also they were used with betel leaves, areca nut and lime which were chewed into a cud and then spat out. But tobacco in the granular form and quids placed

in one part of the oral cavity (Khaini, mawa) has emerged as a major form of tobacco in recent years with alarmingly high usage by younger age groups placing them at risk of chronic exposure. These quids when kept in one particular region of the buccal mucosa for prolonged periods put the overlying mucosa at increased risk for mutagenesis as compared to chewing tobacco which involves exposure of mucosa for a short time after which it is spat out. With this gradual but definitive change in usage pattern, there is a need to reassess the carcinogenic potential and epidemiology of "Indian oral cancer" holistically.

Tobacco flake is one of the major forms of non-smoking tobacco marketed in India, usually with lime as additive. These flakes are made into quids and kept in the oral cavity, particularly in the tobacco alveolar sulcus for varying amount of time. The term used for these flakes varies in different parts of the world (Tobacco quids, khaini in India, moist snuff in United States of America)

With more than 90% of oral cancer attributed to tobacco, the need to curb tobacco usage is more than ever.

This study aims to find the usage pattern of this specific form of tobacco (tobacco quid or Khaini) in patients with oral cancer in comparison with people without cancer.

Various parameters like years of abuse, quantum of use, co-abuse of other substance like bidi, cigarette, alcohol and other smokeless tobacco are assessed.

Despite the advances in medical sciences over the past few decades, the prognosis of Oral squamous cell carcinoma remains dismal with the overall five year survival being 65% for T1,T2 lesions and 30% for more advanced T stages with survival reduced to half these numbers in case of node positivity. This reflects the definite incentive in early diagnosis.

Various techniques like confocal microscopy, Radiolabelled antibodies to tumour markers, narrow band imaging, multi-wavelength fluorescence and reflectance technology are being designed to detect subclinical disease in at–risk patients to identify dysplasia. This translates to a necessity to define the at-risk subset of people among oral tobacco abusers for screening with the above mentioned technique, as screening the entire population of oral tobacco abusers is economically and logistically not feasible.

This study will to an extent try to delineate such high risk population by studying the tobacco use patterns of patients with established oral cancer.

AIMS AND OBJECTIVES

Primary Objectives:

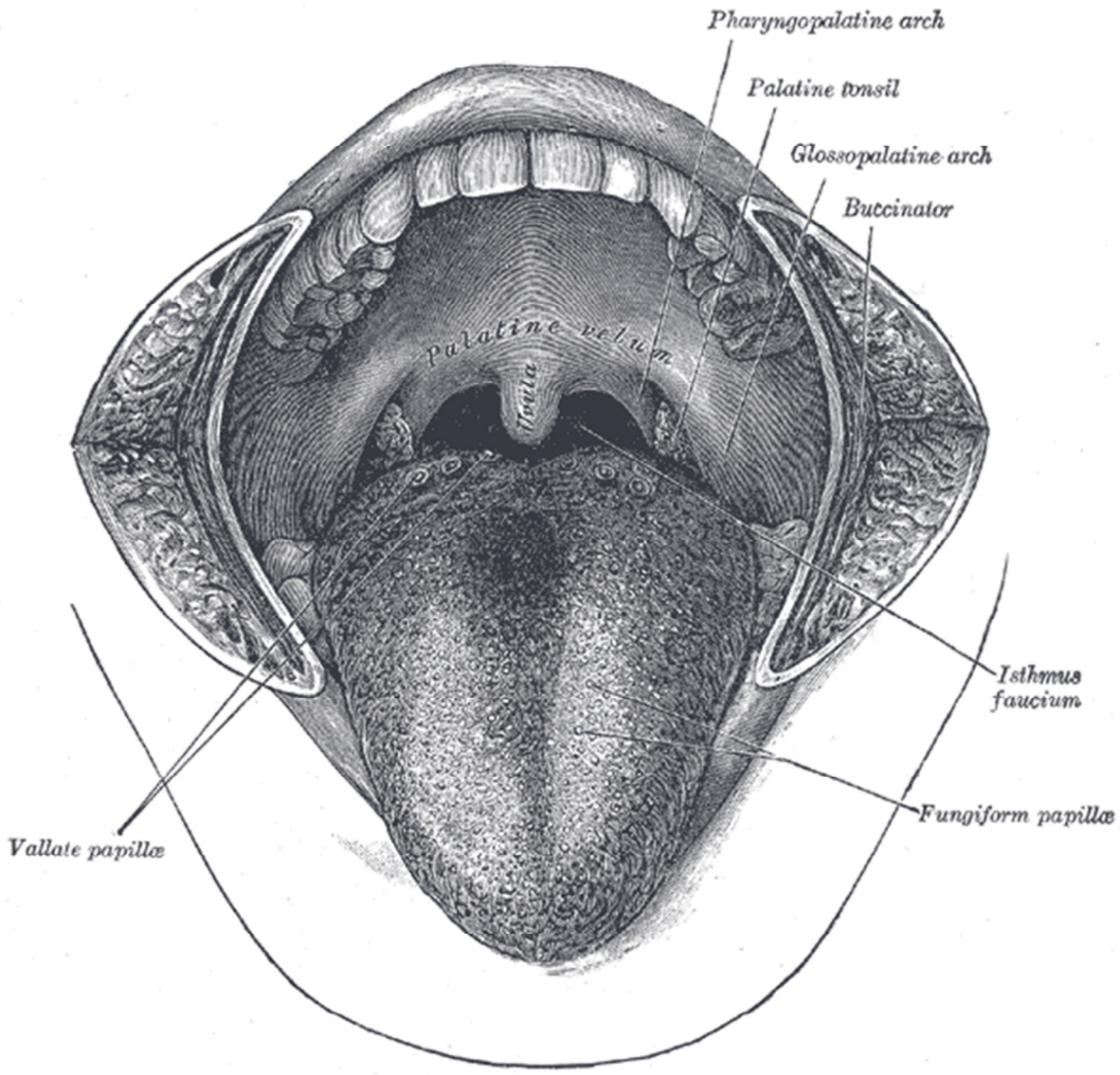
1. To find out mean years of exposure required to produce oral cancer or premalignant lesions
2. To find out specific high risk abuse patterns in patients with oral cancer(overnight abusers, large volume abusers)
3. To find out if the amount of tobacco used per day is significantly more in patients with oral malignancy

Secondary objectives

1. To find out any specific socio economic class with proclivity for high rates of abuse and subsequent high rates of oral malignancy
2. To find out if alcohol and smoking tobacco co-abuse increases the risk of oral cancer.
3. To find out the level of awareness about the ill effects of tobacco among patients with oral cancer
4. Ultimately to serve as a tool to define a high risk subset among people with tobacco quid abuse who are at increased risk of developing oral cancer.

REVIEW OF LITERATURE:

Anatomy of the oral cavity

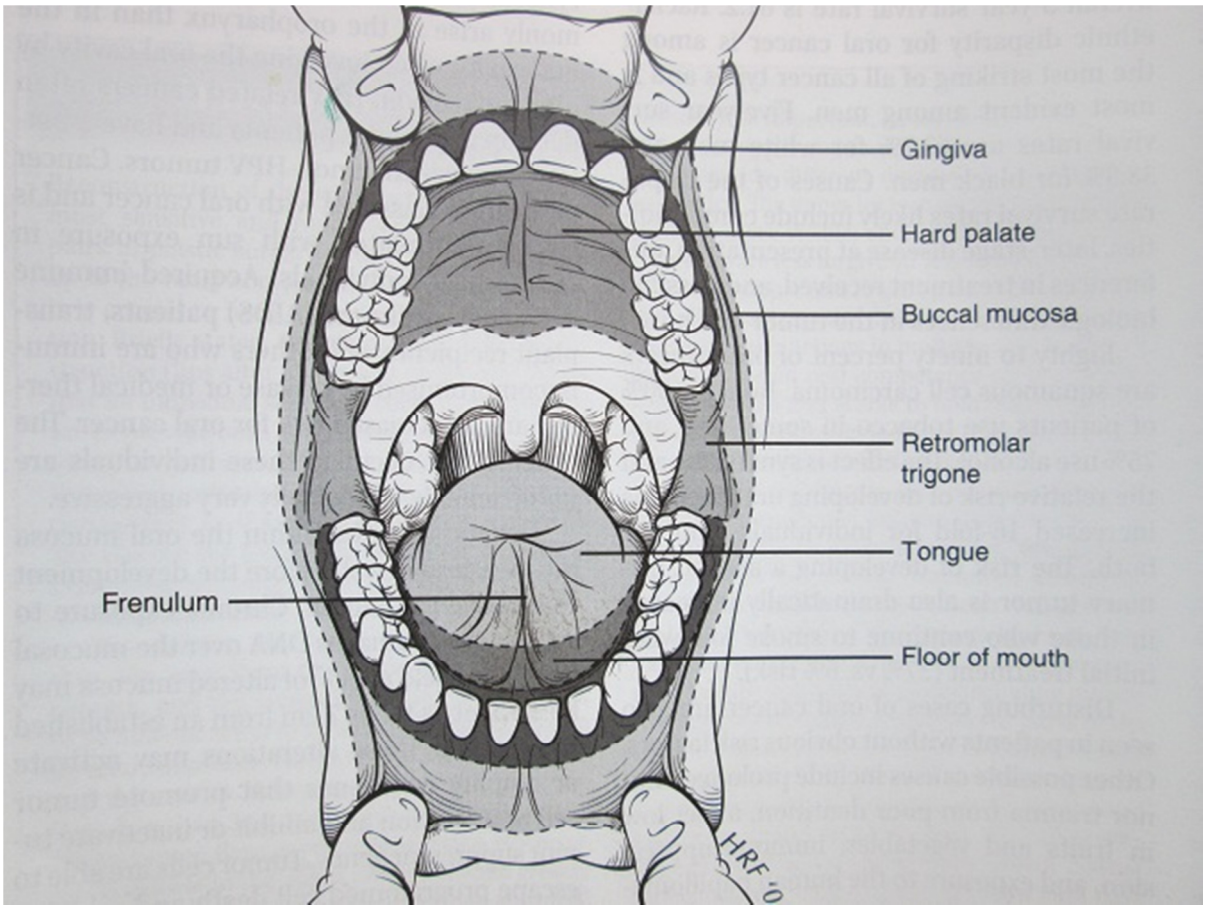


The oral cavity may be subdivided into two smaller cavities: the externally positioned vestibule and the internally placed oral cavity proper.

1. The vestibule is the space bounded by the lips and cheeks anteriorly and laterally, whereas its internal boundary is formed by the dental arches. The ducts of the parotid glands deliver their secretory products into the vestibule
2. The oral cavity proper is bounded by the teeth externally, the floor inferiorly, and the palates superiorly.

At its posterior extent, the oral cavity proper is delineated from the oro-pharynx by an imaginary plane drawn between the palato-glossal folds just anterior to the palatine tonsils.

The oral cavity anatomically includes the lips, buccal mucosa, gingiva, floor of mouth, anterior floor of mouth, anterior two thirds of tongue, hard palate and the retromolar trigone.



ORAL MUCOSA

The epithelium and underlining connective tissue (lamina propria) constitute the oral mucosa. If the epithelium is keratinized (or para keratinized), the mucosa is said to be masticatory mucosa, and if the epithelium is not keratinized, the mucosa is referred to as lining mucosa.

The epithelium consists of four layers of the keratinized oral mucosa and the non-keratinized has the two of deeper four layers but

does not have the two superficial final layer; it has a nonspecific superficial layer instead:

Stratum corneum

Stratum granulosum

Stratum spinosum

Stratum basale

1. Most of oral cavity possesses lining mucosa, with the exception of the gingiva, hard palate, dorsal surface of the tongue, which are covered by masticatory mucosa.
2. The oral cavity has areas of specialized mucosa, located mostly on the dorsal surface of the tongue, though present also on the soft palate and pharynx, where barrel-shaped intraepithelial structures known as taste buds function in taste perception.

Keratinisation is the formation of Stratum corneum from the underlying stratum granulosum. It is not a living layer. The cells of the epidermis arise from the progenitor cells in the stratum basale and differentiate as they move to superficial layers. Non-keratinised epithelium may turn into keratinised epithelium when subjected to chemical injury or trauma. This change is called hyperkeratinisation.

Hyperkeratinisation is more common in the nonkeratinised epithelium. An example is lineal aba which forms in the buccal mucosa in the region where the mandibular and maxillary teeth meet. Hyperkeratinisation slowly reverses if the inciting agent is removed. Hyperkeratinized tissue is also associated with the heat from smoking or hot fluids on the hard palate in the form of nicotinic stomatitis.

The lamina propria is a fibrous connective tissue layer that consists of a network of type I and III collagen and elastin fibers in some regions. The main cells of the lamina propria are the fibroblasts, which are responsible for the production of the fibers as well as the extracellular matrix.

The lamina propria, has two layers: papillary and dense. The papillary layer is the superficial layer, made up of loose connective tissue along with blood vessels and nerve tissue with the tissue comprising an equal amount of fibres, cells, and intercellular substance. The dense layer is the deeper layer of the lamina propria, composed of dense connective tissue with a large amount of fibres. Between the papillary layer and the deeper layers of the lamina propria is a capillary plexus for provision of nutrition to all the layers of mucosa and sends capillaries into the connective tissue papillae.

Depending on the region of the oral cavity a submucosa may or may not be present deep to the lamina propria. The submucosa contains loose connective tissue and may also contain salivary glands.

LYMPHATIC DRAINAGE

Lymphatics from the oral cavity primarily drain into the peri-facial, upper jugular, submandibular and sub-mental nodes and thence to the deep cervical nodes.The most connon sites involved in Oral malignancy are Level I,II,III and IV.

CANCER OF THE ORAL CAVITY

INCIDENCE

Carcinoma of the oral cavity is the 6th most common malignancy in the world accounting for 30% of head and neck malignancies. In India, the scenario is different though with oral cancer being the first and third most common cancers in men and women respectively (1). Though males are more frequently affected than females according to worldwide and India statistics, some studies in India have put the ratio at one(1) implying the wide spread use of tobacco products in India. It has been calculated that the annual incidence of oral cancer in India is between 75,000 and 80,000(2) with usual presentation in the sixth or seventh decade of life.

ETIOLOGY AND RISK FACTORS

TOBACCO

Several carcinogens have been identified in smokeless tobacco. The most important are N-Nitrosamine (TSNA), N-Nitrosonornicotine(NNN), 4-(methylnitrosamino)-1-(3-5pyridyl)-1 butylone(NNK). During curing and fermentation of tobacco (NNK &NNN) are formed in large numbers from nicotine.While in Western countries smoking tobacco makes up the majority of tobacco use, in India a large proportion is accounted for by Smokeless Tobacco.

Familial and Genetic Predisposition

The genealogical records of the Utah (Mormon church) database; show lip to have the strongest familial cancer clustering followed by leukemia, lobular breast cancer, early melanoma, and adenocarcinomas of the lung in females (6). A study from south India revealed the familial association to be less than 1% of the total cancer in oral cavity consistent with autosomal inheritance (5). Copper and his colleagues, (7) in their study on probands of head & neck cancer patients found 31 cases of pulmonary and upper gastrointestinal tract versus 10 cases in the control group comprising of probands of the index patient's partner (n=617) summing upto a 'relative risk' of 3.5 (significant) for first degree relatives, and of 14.6 (significant) for siblings with smoking and drinking histories having no effect, implying a constitutional factor role in the handling of genotoxic substances. Jefferies et al (8) and Llewellyn et al (9) reviewed the evidence in this growing field.

VIRAL INFECTIONS

Viruses play a role in the aetiology of Oral cancers as well. Various viruses have been investigated in this context including Epstein Bar Virus (EBV), Human Simplex Virus-1 (HSV-1) and Human papilloma virus (HPV) types16 and 18 (10). The strongest association is between HPV and squamous cell carcinoma of the oral cavity, more so in the

oropharynx (11). There is sufficient evidence for a causal role of HPV-16 in this context but it is limited for HPV-18 (12). These viruses are found in approximately 25% of cases in India and 85.7% of cases in Taiwan (13). In Taiwan high risk HPV (Subtypes 16 and 18) emerged as independent risk factor even after adjusting for age, gender, cigarette smoking and low risk HPV infection. The OR for OC due to HPV-16 (11.21) is greater than that for HPV-18 (13). In a Malaysian population, the OR was lower (4.3) (15). HPV is less common in cases that chewed quid or smoked when compared to those who had multiple sexual partners (13). Contracting HPV infection by practising oral sex is more likely in men and increases the risk of OC by 3 fold (14). HPV acts synergistically with betel quid chewing to cause high morbidity (13). HPV-16 infection is more common than HPV-18.

Immunisation against HSV has been found to be carcinogenic due to the effect of dimethylbenzathracene. This may imply that herpes simplex virus may act as a co-carcinogen with various other carcinogens like tobacco and other substances. It has been postulated that there is generalised immunosuppression induced by smoking particularly of the NK cells activity, thereby favouring chronic HIV infections and carrier state which can lead to raised antibody titre(16) explaining high titres of HSV antibodies in smokers and those with head and neck cancer.

FUNGAL INFECTIONS

Fungal invasion of leukoplakia is seen in some cases especially in nodular variety (17). This carries tremendous risk for transforming into malignant lesion. The link between smoking and candida infection is well established and is similar to the risk in HIV patients (17). Iron deficiency state predisposes to oral cancer as well as oral candidiasis. It seems that candidiasis per se does not cause oral malignancy but most probably is involved when multiple other causative factors are present. Enzymes present in candida causes nitrosation of food substances and this may lead to carcinogensis.

DENTAL FACTORS

The clear association between poor oral hygiene and oral cancer has been well established. In addition to this there is also clear association between sharp, fractured teeth, prosthesis (18). But in most cases it is difficult to attribute a causal role to the above said factors because of the presence of confounding factors like nutrition, alcohol, tobacco abuse and socio-economic status. Further well controlled studies are needed to establish the role of these factors. Previous studies have demonstrated

that a tumor may arise from the site of chronic ulceration caused by a broken tooth or dental prosthesis and this is the probable mechanism by which the above said factors may facilitate transformation.

PATHOGENESIS

Though the carcinogenic potential of smokeless tobacco has been well established, the genetic abnormalities and premalignant conditions differ significantly from that caused by smoking tobacco. In cases of smoking tobacco, long before the development of invasive cancer, genetic changes occur within the cells of the oral mucosa which are measurable. Chronic exposure to carcinogenic material damage the DNA over the mucosal field .This altered mucosa can extend as far as 7 cm from the site of malignancy. These molecular alterations may include amplification of oncogenes that promote tumor cell proliferation along with inactivation of tumor suppressor genes. This enables tumor cells to escape programmed cell death, grow and divide in a self-sufficient manner.

Most of the oral premalignant lesions (70-80%) have changes in 9p21 chromosome which encodes for tumor suppressor genes p16 and

p14. These genes are inactivated by the epigenetic process of methylation.

P53 mutation in oral malignancy has received much attention recently but it is a late occurrence in the transformation process. P53 mutation in the margins of resected tissue during surgery correlates with increased rate of recurrence in the future.

PREMALIGNANT LESIONS

LEUKOPLAKIA

Leukoplakia is defined as “a white plaque that does not rub off and cannot be clinically identified as any other entity”. Not all leukoplakias are premalignant lesions. Dysplastic changes are seen in a meagre 20% population. But in some sites (floor of mouth, under surface of tongue) this is as high as 45%. There is no reliable way to correlate the clinical appearance of leukoplakia and the presence of dysplastic change detected histologically. If the leukoplakia is interspersed with erythroplakic component the risk of malignant transformation increases tremendously, termed as “erythro-leukoplakia”.

ERYTHROPLAKIA

An erythroplakia is defined as “a red lesion that cannot be classified as a clinical entity”. In contrast to leuokoplakia, there is a very high chance of dysplasia or malignancy in this condition approaching 90%. There may be foci of keratosis which appear as speckled white spots in a background of flat red velvet appearance.

LICHEN PLANUS

It is still disputed that lichen planus lesion is a premalignant lesion. It is a relatively common lesion and it is believed by some that the dysplasia or carcinoma found in patients with lichen planus may be coincidental. However many studies (Oral Cancer Background Papers) have reported high percentage of malignant transformation in patients with lichen planus. Since the malignant transformation is disputed, it would be prudent clinical practice to biopsy the lesion at the initial clinical presentation and monitor the patient for premalignant and malignant changes.

MUCOSAL LESIONS ASSOCIATED WITH QUID

Quid chewing habits are associated with specific oral mucosal lesion which can be classified into 2 broad categories:

1. "Lesions or conditions that are diffusely outlined, involve more than one site or represent a widespread alteration, such as those due to mechanical or chemical trauma".

2. "Lesions that are localized to the site where quid is regularly placed. These lesions are equivalent to snuff-induced lesions or tobacco–lime user's lesions, which arise only on the mucosa in contact with the quid".

BETEL CHEWER'S MUCOSA

Because of the traumatic effect of chewing or the direct action of betel quid there is a tendency for oral mucosa to desquamate and peel. Sometimes white tags of loose, detached tissues can be seen and also felt. The area underlying these loose tissues will have wrinkled or pseudomembranous appearance. There may also be yellow or reddish brown encrustations denoting the incorporation of ingredients of quid in the mucosa. Cheek-biting will also lead to a very similar appearance both

clinically and histologically which should be differentiated from betel chewer's mucosa. It should be noted that betel chewer's mucosa results from an intentional habit while cheek biting is unintentional. Also cheek biting lesions occur in younger most likely in their 3rd or 4th decade of life while betel chewer's mucosa occurs in those aged 50 and above.

QUID-INDUCED LESION

This lesion occurs in the site of the oral mucosa where the quid is placed regularly. It is characterised by the following: presence of ulcers, thickened mucosa, colour change, scrapable/non-scrapable epithelial surface and wrinkled appearance.

ARECA-NUT-RELATED LESION

This lesion in characterised by a greyish white discolouration which cannot be rubbed off. Histologically there may be para-keratinised or ortho-keratinised epithelium.

PROBABILITY OF MALIGNANT CHANGE

The percentage of epithelia dysplasia becoming malignant is 5-18. Some types of epithelial dysplasia show high chances of malignant

transformation than others but recognising the types of epithelial dysplasia with malignant change has proved difficult because most cases are excised, instead of being followed up for malignant change. Epithelial dysplasia with high malignant change may show the following changes:

(1) An erythroplakia arising from within a leukoplakia,

(2) verrucous appearance of the lesion

(3) Lesions located in anatomic site such as the tongue or floor of mouth where most cases of oral cancer occur

(4) Negative history for smoking

(5) More than one lesion.

TIME TAKEN FOR EPITHELIAL DYSPLASIA TO UNDERGO MALIGNANT CHANGE

Even though epithelial dysplasia is present around the regions of frank malignancy, not all arise from these areas, few may arise de novo from cells in the stratum basale. Silverman and colleagues (2) in their study noted the following “out of 257 patients with oral leukoplakia; 22 had a diagnosis of epithelial dysplasia, the remaining 235, hyperkeratosis.

Eight of the 22 (36.4%) with epithelial dysplasia developed carcinoma. Of the 107 patients with a homogeneous leukoplakic lesion and a diagnosis of hyperkeratosis, 6.5% developed carcinoma. However, 23.4% of the 128 patients with erythroplakic lesions and a diagnosis of hyperkeratosis were eventually diagnosed with carcinoma. The time from initial diagnosis of either epithelial dysplasia or hyperkeratosis to carcinoma ranged from 6 months to 39 years". In another study reported by Lumerman and colleagues, "15.9% of 44 patients with oral epithelial dysplasia identified in a biopsy service developed carcinoma; the mean time from biopsy to cancer diagnosis was 33.6 months. Epithelial dysplasia has been more extensively studied in association with the uterine cervix than with the oral cavity. Based on clinical reviews, approximately 12% of cervical epithelial dysplasias progress to carcinoma in situ. The estimated median time for this progression depends on the histologic severity of the epithelial dysplasia: 58 months for mild, 38 months for moderate, and 12 months for severe. Approximately 73% of carcinoma in situ cases evolve into full-blown carcinoma. The importance of this information in understanding the progression to oral cancer is unclear, but it is consistent with observations that not all oral epithelial dysplasia's evolve into carcinoma in situ or full-blown carcinoma and that this transition—when it does occur—takes months or years".

STAGING

Staging of Oral malignancy is based on the TNM system according to the 7th edition (2010) American Joint Committee on Cancer (AJCC) Guidelines

Primary Tumour (T)

TX Primary cannot be assessed

T0 No evidence of primary tumor

T1 Tumor not more than 2cm in greatest diameter

T2 Tumour between 2 cm and 4 cm in its greatest diameter

T3 Tumor greater than 4 cm in greatest dimension

T4 Tumour invades adjacent structures (cortical bone, deep intrinsic muscles of tongue, maxillary sinus or skin)

REGIONAL LYMPH NODES (N)

N0 No regional lymph nodes

N1 Metastasis to single ipsilateral lymph node, 3cm or less in greatest dimension

N2a Metastasis to single ipsilateral lymph node >3cm but <6cm

N2b Metastasis to multiple ipsilateral lymph nodes none >6cm

N2c Metastasis to bilateral or contralateral lymph nodes none > 6cm

N3 Metastasis in a lymph node > 6cm

DISTANT METASTASIS (M)

MX Distant metastasis cannot be assessed

M0 No distant metastasis

M1 Distant Metastasis

STAGE GROUPING

Stage I T1, N0, M0

Stage II T2, N0, M0

Stage III T3, N0, M0 or T1-3, N1, M0

Stage IV T4, N0-1, M0 or Any T, N2-3, M0 or Any T, Any N, M1

The TNM classification doesn't take into account the depth of the tumour which several clinicians feel correlates with the occurrence of nodal metastasis. A depth of four millimetres or more is said to be significant.

TREATMENT

Optimal treatment requires a multidisciplinary team including head & neck surgeons, reconstructive surgeons, medical oncologist and radiation oncologist. Speech and swallowing pathologists rehabilitate functions lost during multimodal therapy. Other important team members include dentists, prosthodontist, nutritionist and social workers. A multi-specialty tumour board needs to be considered for advanced and recurrent cases.

TREATMENT SELECTION FOR THE PRIMARY SITE: GENERAL PRINCIPLES

For early cancer of oral cavity, either radiotherapy or surgery is preferred. When comparing the cure rates of the above mentioned

modalities there is no significant difference. The selection of either surgery or radiotherapy depends on a large number of factors like availability of specialised surgical oncology units, the surgeon's philosophies, patient personal preference, general condition, complication risk, required functional outcome, the cost and length of treatment.

For more advanced lesions, combined radiotherapy and surgery is preferred. Post-operative radiotherapy is preferred now-a-days over pre-operative radiotherapy because surgical complications are higher if the primary had been irradiated previously. Common indications for post-operative radiotherapy of the primary includes

- Margin positivity
- Peri-neural invasion
- Vascular invasion

T1AND T2 CARCINOMA OF THE ORAL CAVITY

Single modality treatment is adequate for T1, T2 carcinoma of the oral cavity. Whichever modality is chosen, treatment of the primary site and neck should be consistent. In planning resection the route of access,

margin status, bone involvement and whether or not lymphatics require treatment should be considered. Bone involvement may require segmental mandibulectomy or at least a rim mandibulectomy .

T3 AND T4 CARCINOMA OF ORAL CAVITY

Multimodal therapy is usually required for T3 and T4 squamous cell carcinoma of oral cavity. Soft tissue defects are usually repaired with radial forearm, lateral thigh or rectus abdominus flap. Mandibular defects are managed with ostio cutaneous flap, the fibular flap being the most common.

GENERAL PRINCIPLES OF MANAGING THE SECONDARIES IN THE NECK

It is well known that the incidence of secondary cervical nodal deposit increases with increasing stage of disease. For early stage of oral cancer, the neck is treated with the same modality as the primary (RT or surgery). Sometimes when the risk of secondary deposition in the neck is low (15%), the neck can simply be observed for occurrence of nodal metastasis.

For more advanced diseases of the neck, combined modality treatment is used. Indications of pre-operative neck radiotherapy includes

- Large nodal mass
- Fixity to carotid artery
- Fixity to other adjacent structures

Indications for post-operative neck radiotherapy includes

- Multiple node positivity
- Extra nodal involvement

CHEMOTHERAPY

Even though radiotherapy and surgery can cure most early stage diseases, the prognosis when it comes to advanced diseases is dismal. In these advanced stages, chemotherapy is sometimes used to reduce distant metastasis and improve survival rates. The role of chemotherapy in these circumstances is still debatable.

Neo-adjuvant chemotherapy with combination of 5-fluorouracil and cisplatin showed response rate ranging from 60-90% in some studies. Around 20-40% showed complete clinical response (patients who experienced a complete clinical response have a better prognosis than

people with partial or no response). But in multiple randomised control trials increase in survival rates could not be demonstrated.

Concomitant chemo-radiation for loco-regional disease control has resulted in increased disease free survival and better regional control in several well randomised control trials. But the most important adverse effect of this modality is the significant toxicity seen in many patients.

Adjuvant chemotherapy has yielded very discouraging results and its use is no longer recommended in the treatment of squamous cell carcinoma of oral cavity.

NEWER TRENDS IN THE MANAGEMENT OF ORAL CAVITY

Monoclonal antibodies targeted at individual tumor cells are being used to deliver chemotherapeutic agents to the tumour. This will ensure high local tumor concentration of the chemotherapeutic agent and at the same time decreasing the chances of systemic toxicity. Added to this, monoclonal antibodies can be tagged with radiolabelled materials for a better and improved imaging of the tumor. Immunomodulators like alpha interferons and interleukin have been tried.

Stereotactic radiosurgery, a new and emerging modality is being tried on those who had shown inadequate or no response to surgery/radiotherapy. Newer radiotherapy techniques like twice-daily radiotherapy, accelerated fractioned radiotherapy are being tried and these have been found to be useful in stage II & III oral malignancies.

Gene therapy which involves introducing new genetic material into the human DNA in an attempt to inhibit oncogenes, activate tumor suppressor genes, activate host immune response, make the tumor more sensitive to chemo and radiotherapy. Even though there are no concrete results so far, the future of gene therapy in oral cancer remains promising.

Regarding surgery, the use of osseo-integrated dental implants have paved way for more aggressive tumor removal without compromising on oral function. Neural micro-anastomosis after free flap have been tried to improve functional outcome. Micro-vascular free flap technique have led to better outcomes following large free flap transfers.

PROGNOSIS:

5 years survival rate range from 85% to 90% for stage1 and 70% to80% for stage 2 squamous cell carcinoma of the oral cavity.

For T3 and T4 squamous cell carcinoma of the oral cavity, the survival rate is 50% to 65%. In the case of nodal metastases the survival drops to half of the above mentioned value (25-30%).

METHODS AND METHODOLOGY

100 Patients with established oral cancer and tobacco quid abuse were chosen and compared with age matched disease-free users of tobacco quid.

The period of study was from May 2012 to December 2013.The cases were chosen from in-patients and out-patients attending the departments of Radiation oncology, surgical oncology and General surgery. A total of 372 cases of oral cancer were identified during the study period, of which 100 cases were selected after applying inclusion, exclusion criteria. Stage of the disease was found out from the case sheets in case of inpatients and medical records in case of out-patients. The selected subjects were subject to a semi-structured oral questionnaire about their non-smoking tobacco usage which included detailed questions about the number of times used per day, years of abuse and other substance abuse after obtaining informed consent. The patients were also provided with information sheets about the study in their native language.

Controls were chosen from inpatients in the department of General surgery who were admitted for ailments other than malignancy. Controls were also chosen from attenders of patients who came as visitors to the hospital and were willing to take part in the study. The cases and controls were matched for of age. They were issued patients information sheets in their native language and informed consents was obtained in writing .The

controls were subjected to similar oral questionnaire as was used for the cases. In addition the controls were subjected to a clinical oral examination to exclude any un-diagnosed malignant or pre-malignant lesions.

INCLUSION CRITERIA:

Cases:

1- People with histologically proven squamous muscle carcinoma of the oral cavity with history of tobacco quid (smokeless tobacco) abuse.
2- All ages

Control:

1- Any person with history of tobacco quid abuse with no history of present or past malignancy.
2- All ages

EXCLUSION CRITERIA:

Cases:

1- Patients with established squamous muscle carcinoma of the oral cavity with no history of past or present Tobacco quid abuse (smokeless tobacco)
2- Unstable physical condition
3- Non-consenting subjects

Controls:

1- People with history of past or present malignancy anywhere in the body

2- People found to have oral malignant or premalignant lesions on clinical examination

2- Non-consenting individuals

DATA COLLECTION

Data collection was done with a predetermined oral semi-structured questionnaire.

DATA COLLECTION SHEET

Name:

Age: Sex: IP/OP no:

Department:

Diagnosis:

Stage:

Education:

1. Professional 2.Graduate 3.Diploma

4. High School 5.Middle School 6.Primary school

7. Illiterate

Profession

1. Professional 2.Semiprofesional 3.Clerical/farmer

4. Skilled 5.Semi-skiled 6.Unskilled

7. Unemployed

Years of Exposure:

No of Quids/day:

Overnight use: 1.Never 2.Ocassionally 3.Regularly

Average time per Quid: 1.Less than half an hour

2. Half an hour to one hour

3. More than one hour

Substance	1.Never	2.Occasionally	3.Regularly
BIDI			
CIGARETTE			
ALCOHOL			
OTHER ST			

Awareness about ill effects:

History of abstinence:

Educational status and profession were based on the modified Kuppusamy scale. Abstinence is said to be present if the patient or control had abstained for one year or more preceding the time of interview.

STATISTICAL ANALYSES:

The data obtained was subject to statistical analysis using SPSS. Categorical variables were analysed with chi-square test, Continuous variables with t-test and Odd's ratio was computed for relevant variables.

RESULTS

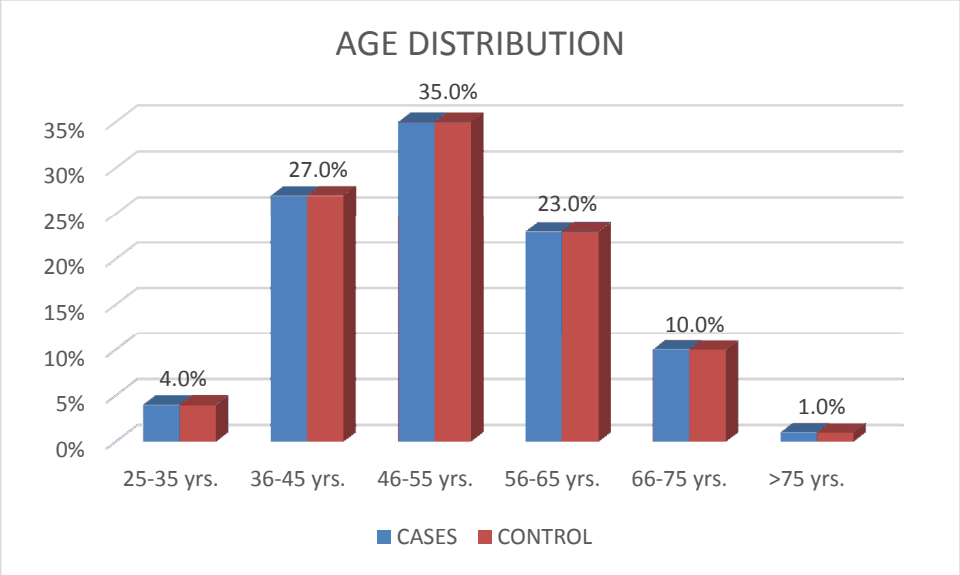

AGE DISTRIBUTION
35%
30%
25%
20%
15%
10%
5%
0%
4.0%
27.0%
35.0%
23.0%
10.0%
1.0%
25-35 yrs.
36-45 yrs.
46-55 yrs.
56-65 yrs.
66-75 yrs.
>75 yrs.
CASES
CONTROL

TABLE -1: AGE DISTRIBUTION

Age group	Cases	Control
	%	%
25-35 yrs.	4	4
36-45 yrs.	27	27
46-55 yrs.	35	35
56-65 yrs.	23	23
66-75 yrs.	10	10
>75 yrs.	1	1

The most common age group affected by Squamous cell carcinoma of the oral cavity is between 46-55 years accounting for 35% of cases. Thirty one percent of the cases occurred in patients below the age of 45 years.

TABLE-2: AGE – MEAN COMPARISON

GROUP	Mean age
Case & Control	51.15

The mean age at presentation is 51.15 years

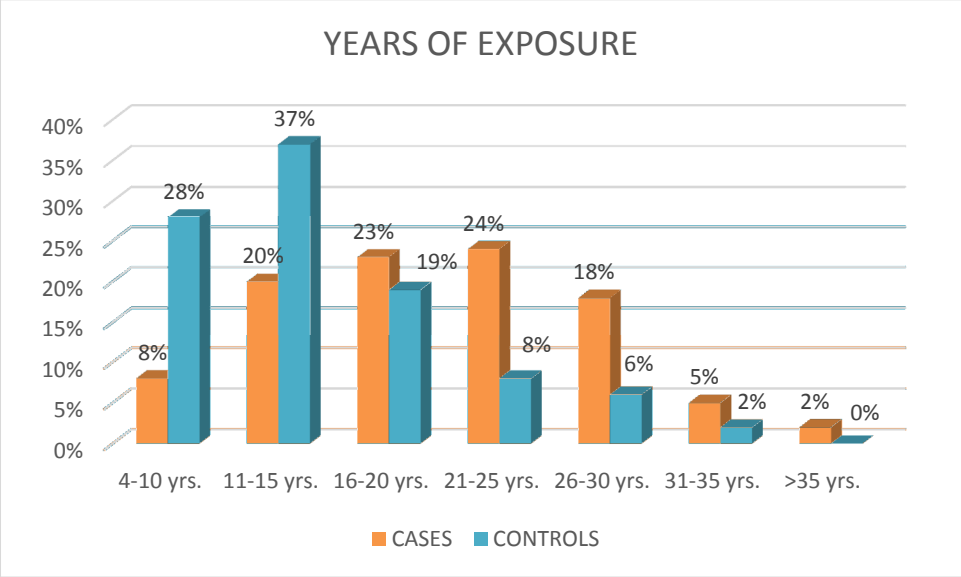

YEARS OF EXPOSURE
40%
35%
30%
25%
20%
15%
10%
5%
0%
8%
28%
20%
37%
23%
19%
24%
8%
18%
6%
5%
2%
2%
0%
4-10 yrs.
11-15 yrs.
16-20 yrs.
21-25 yrs.
26-30 yrs.
31-35 yrs.
>35 yrs.
CASES
CONTROLS

TABLE-3: YEARS OF EXPOSURE

Years of exposure	Cases		Control		Total	
	N	%	N	%	N	%
4-10 yrs.	8	8	28	28	36	18
11-15 yrs.	20	20	37	37	57	28.5
16-20 yrs.	23	23	19	19	42	21
21-25 yrs.	24	24	8	8	32	16
26-30 yrs.	18	18	6	6	24	12
31-35 yrs.	5	5	2	2	7	3.5
>35 yrs.	2	2	0	0	2	1
Chi square value	33.8480					
Df	6					
P- value	**0.00 (Significant)**					

65% percent of controls were exposed to smokeless tobacco for less than fifteen years. 65% of cases were exposed to smokeless tobacco for more than fifteen years and 35% percent of cases were exposed for fifteen years or less.

Of note is the 8% of cases who were exposed for ten years or less.

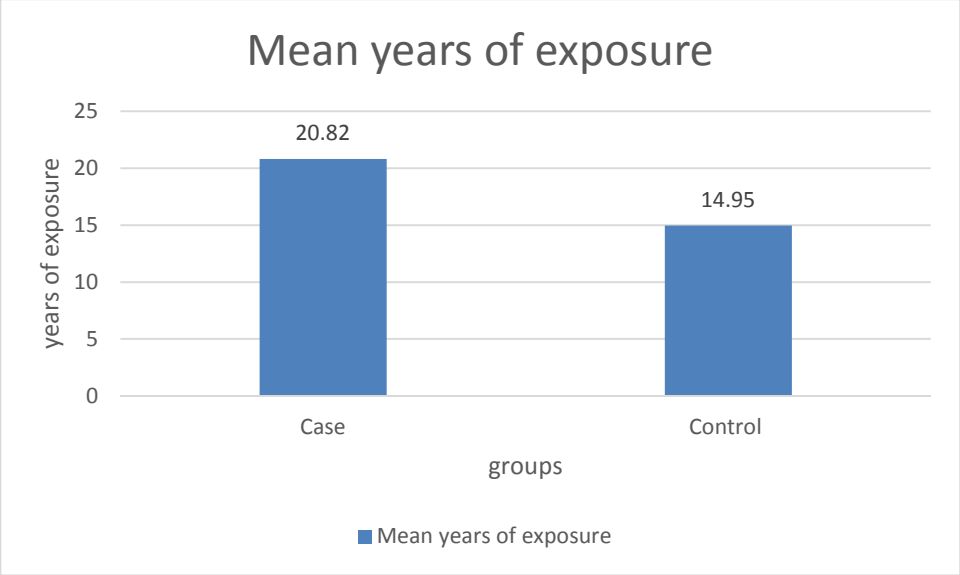

Mean years of exposure
25
20
15
10
5
0
years of exposure
20.82
14.95
Case
Control
groups
Mean years of exposure

TABLE -5: MEAN YEARS OF EXPOSURE COMPARISON

GROUP	Mean years of exposure	S.D	T-test	P-value
Case	20.82	7.06	6.20	**0.00 (sig.)**
Control	14.95	6.28		

The mean years of exposure in cases was found to be 20.82 years with a Standard Deviation of 7.06 years. While for the age matched controls, the mean years of exposure was 14.95 years with a Standard Deviation of 6.28 years.

This difference is statistically significant (**p-0.00**)

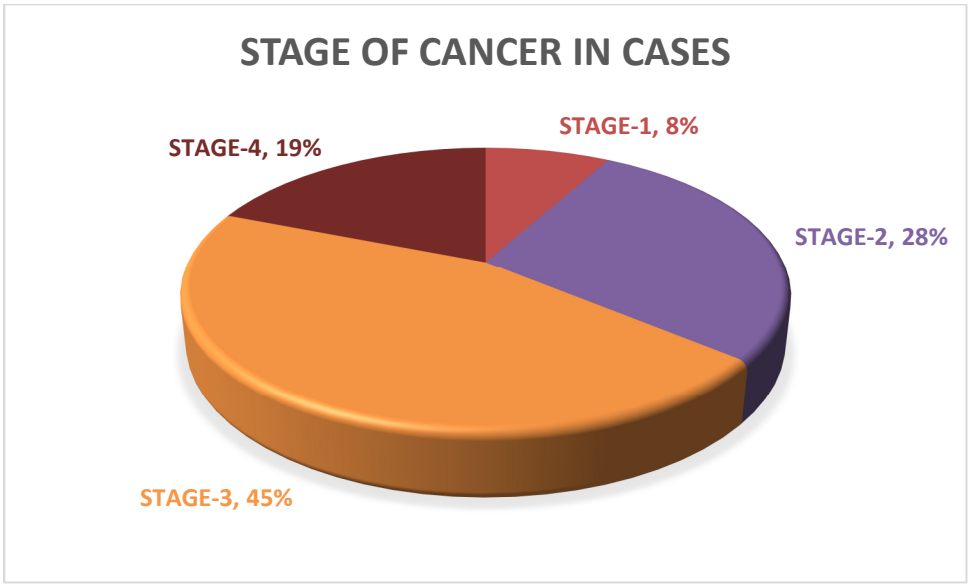

STAGE OF CANCER IN CASES
STAGE-1, 8%
STAGE-2, 28%
STAGE-3, 45%
STAGE-4, 19%

TABLE-6: STAGING OF CANCER

GROUP	Staging of cancer			
	1	2	3	4
Cases	8	28	45	19
Total %	8%	28%	45%	19%

45% of patients presented with stage III disease.

A total of 64% of patients presented with stage III disease and above.

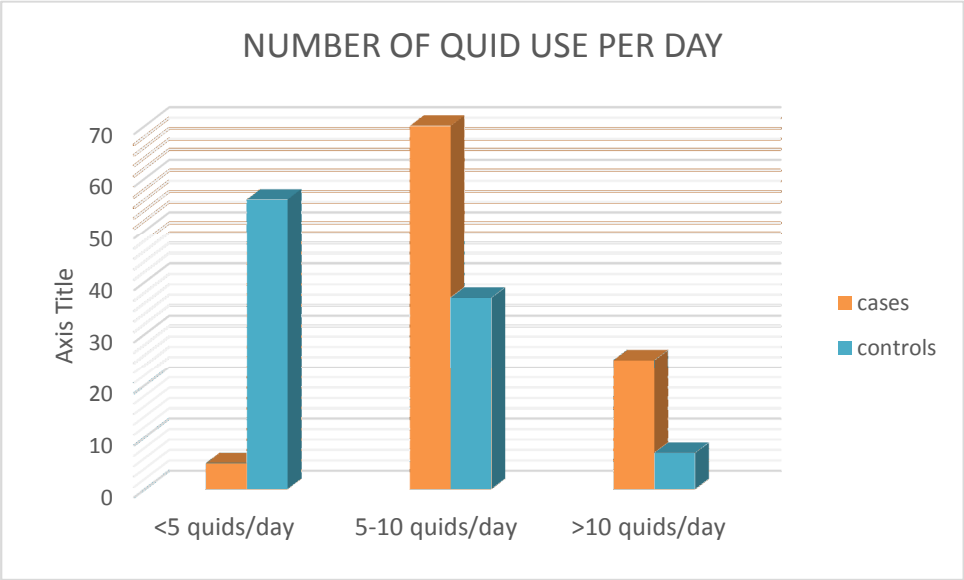

NUMBER OF QUID USE PER DAY
Axis Title
70
60
50
40
30
20
10
0
<5 quids/day
5-10 quids/day
>10 quids/day
cases
controls

TABLE-7: NUMBER OF QUIDS PER DAY USAGE

Group	Number of quid's per day		
	<5	6-10	>10
Cases	5	70	25
Controls	56	37	7
Total	61 (30.5%)	107 (53.5%)	32(16%)
Chi square value	62.9419		
Df	2		
P- value	**0.00 (significant)**		

95% of cases were moderate (6-10 quids/day) to heavy users (>10 quids/day). Only 5% of cases were light users (<5 quids/day). In contrast, 56% of age matched controls were light users. Only 7% of controls were heavy users.

This difference was found to be statistically significant (**P-0.00**)

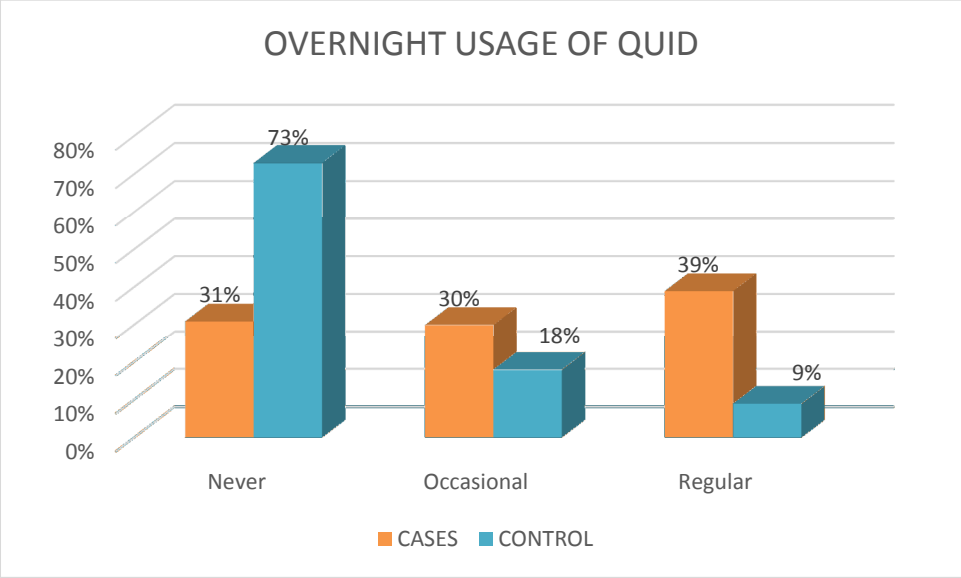

OVERNIGHT USAGE OF QUID
80%
70%
60%
50%
40%
30%
20%
10%
0%
31%
73%
30%
18%
39%
9%
Never
Occasional
Regular
CASES
CONTROL

TABLE- 8: OVERNIGHT USAGE

Overnight use	CASES		CONTROL		TOTAL	
	N	%	N	%	N	%
Never	31	31	73	73	104	52
Occasional	30	30	18	18	48	24
Regular	39	39	9	9	48	24
Chi square value	38.7115					
Df	2					
P- value	**0.00 (significant)**					

Thirty nine percent of cases regularly used tobacco quid in their mouth during sleep, as against only nine percent of controls.

Thirty percent of cases accept occasional use of quids during the night as opposed to only eighteen percent of controls.

Thirty one percent of cases have never used quids overnight as against seventy three percent of controls.

This difference is statistically significant (**p-0.00**).

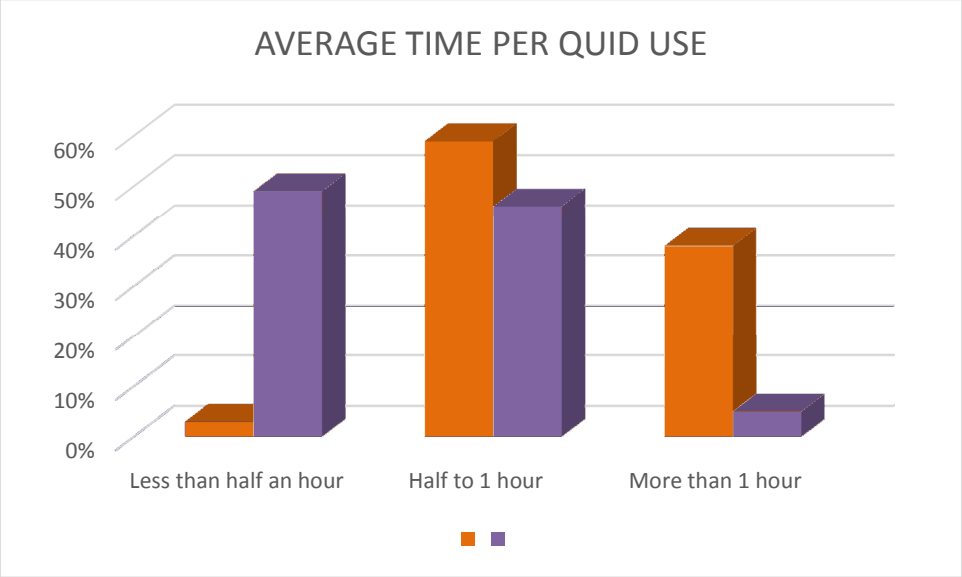

AVERAGE TIME PER QUID USE
60%
50%
40%
30%
20%
10%
0%
Less than half an hour
Half to 1 hour
More than 1 hour

TABLE-9: AVERAGE TIME PER QUID USE

Average time per quid	CASES		CONTROL		TOTAL	
	N	%	N	%	N	%
Less than half an hour	3	3	49	49	52	26
Half to 1 hour	59	59	46	46	105	52.5
More than 1 hour	38	38	5	5	43	21.5
Chi square value	67.6274					
Df	2					
P- value	**0.00 (significant)**					

59% of cases kept quid in oral cavity for half to 1 hour, 38% for > 1 hour. In controls, 49% used for < half an hour, 46% for half to 1 hour. This was statistically significant.

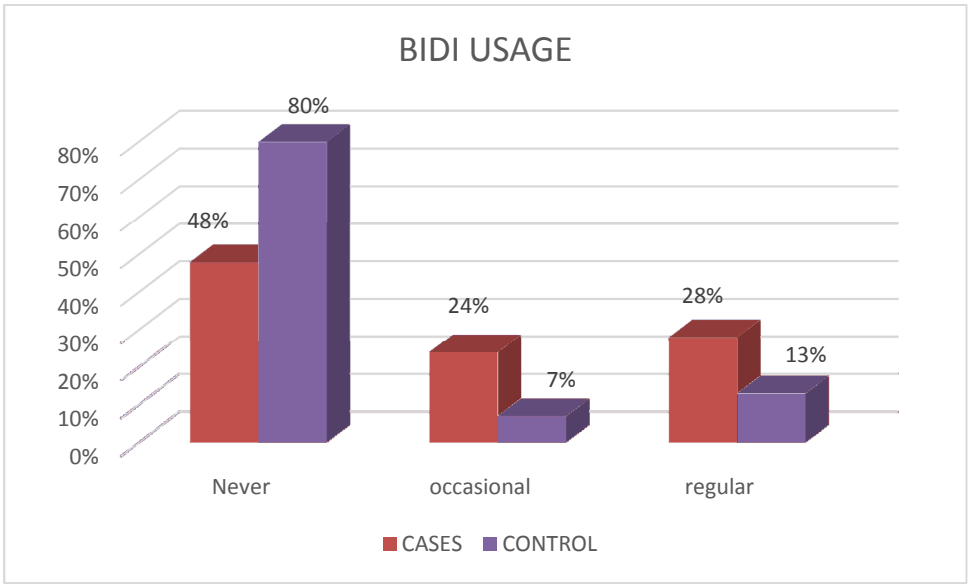

BIDI USAGE
80%
70%
60%
50%
40%
30%
20%
10%
0%
48%
80%
24%
7%
28%
13%
Never
occasional
regular
CASES
CONTROL

TABLE-10: BIDI USE

Bidi use	CASES		CONTROL		TOTAL	
	N	%	N	%	N	%
Never	48	48	80	80	128	64
occasional	24	24	7	7	31	15.5
regular	28	28	13	13	41	20.5
Chi square value	22.8104					
Df	2					
P- value	**0.00 (significant)**					

There was 80% non-bidi user in control as compared to 48% in cases and regular users were higher in cases (28%) than in controls (13%). This was statistically significant.

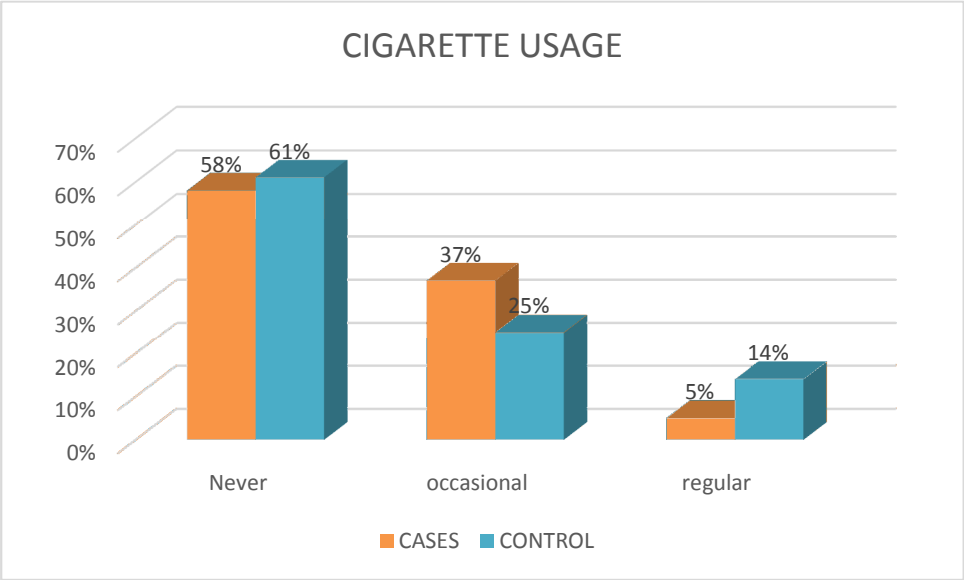

CIGARETTE USAGE
70%
60%
50%
40%
30%
20%
10%
0%
58%
61%
37%
25%
5%
14%
Never
occasional
regular
CASES
CONTROL

TABLE-11: CIGARETTE USE

Cigarette use	CASES		CONTROL		TOTAL	
	N	%	N	%	N	%
Never	58	58	61	61	119	59.5
occasional	37	37	25	25	62	31
regular	5	5	14	14	19	9.5
Chi square value	6.6614					
Df	2					
P- value	**0.03 (significant)**					

61% of controls were non-cigarette users, 25% occasional and 14% regular users. In cases, 58% were non users, 37% occasional and 5% regular users. This difference was statistically significant.

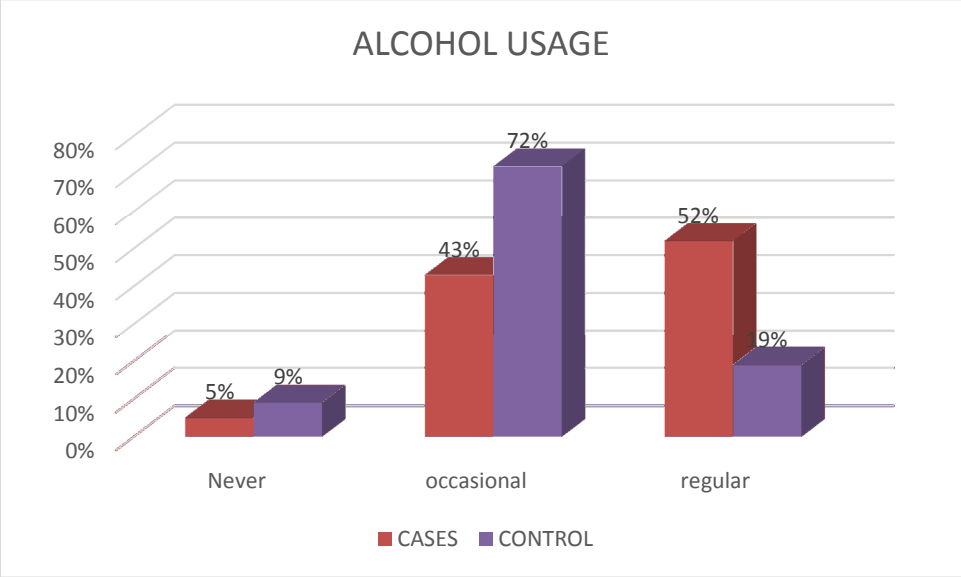

ALCOHOL USAGE
80%
70%
60%
50%
40%
30%
20%
10%
0%
5%
9%
43%
72%
52%
19%
Never
occasional
regular
CASES
CONTROL

TABLE-12: ALCOHOL USE

Alcohol use	CASES		CONTROL		TOTAL	
	N	%	N	%	N	%
Never	5	5	9	9	14	7
occasional	43	43	72	72	115	57.5
regular	52	52	19	19	71	35.5
Chi square value	23.7939					
Df	2					
P- value	**0.00 (significant)**					

While 72% of controls and 43% of cases were occasional alcohol users, 19% of controls and 52% of cases were regular alcohol users. This difference was statistically significant.

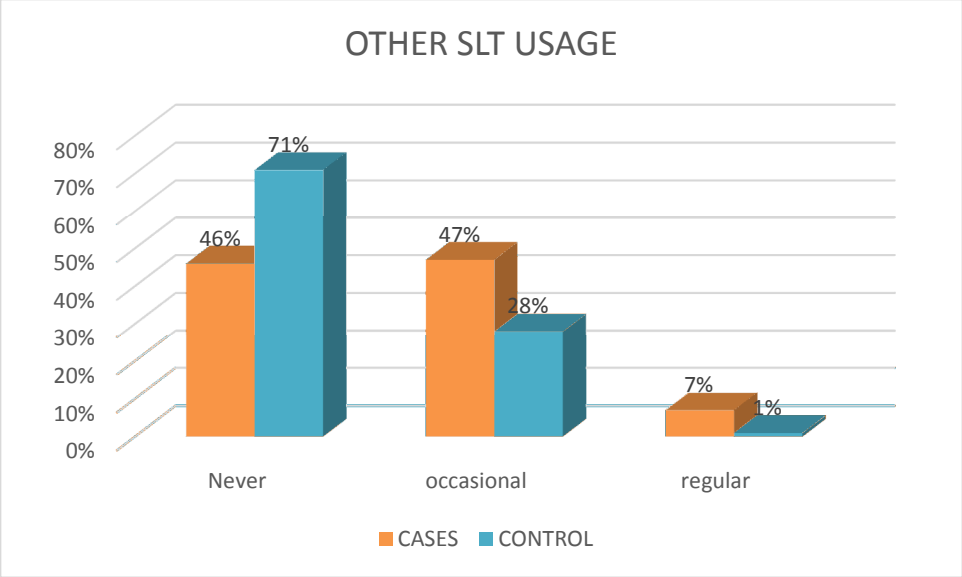

OTHER SLT USAGE
80%
70%
60%
50%
40%
30%
20%
10%
0%
46%
71%
47%
28%
7%
1%
Never
occasional
regular
CASES
CONTROL

TABLE -13: OTHER SLT USE

Other SLT use	CASES		CONTROL		TOTAL	
	N	%	N	%	N	%
Never	46	46	71	71	117	58.5
occasional	47	47	28	28	75	37.5
regular	7	7	1	1	8	4
Chi square value	14.6552					
Df	2					
P- value	**0.00 (significant)**					

The % of SLT was significantly higher than in cases

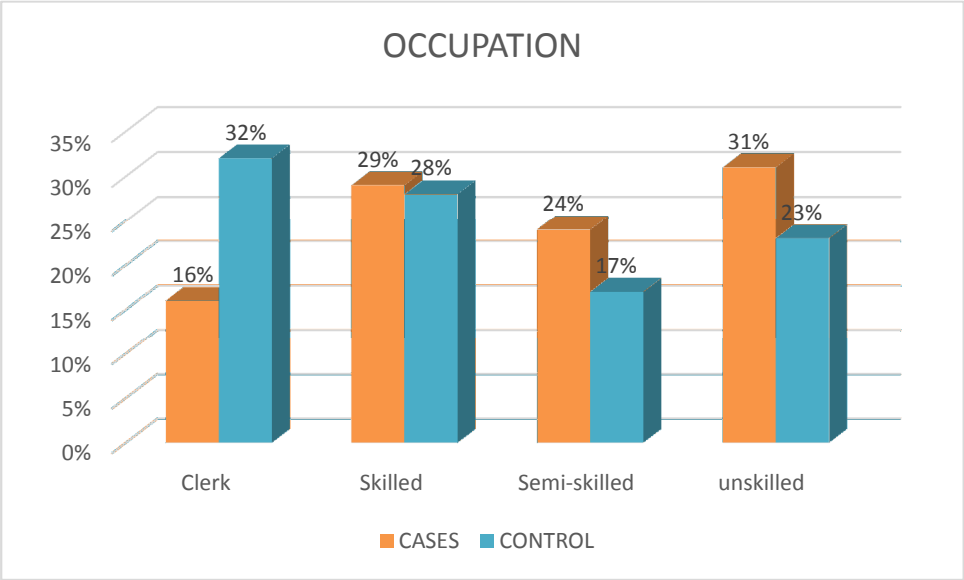

OCCUPATION
35%
30%
25%
20%
15%
10%
5%
0%
16%
32%
29%
28%
24%
17%
31%
23%
Clerk
Skilled
Semi-skilled
unskilled
CASES
CONTROL

TABLE-14: OCCUPATION

Occupation	CASES		CONTROL		TOTAL	
	N	%	N	%	N	%
Clerk	16	16	32	32	48	24
Skilled	29	29	28	28	57	28.5
Semi-skilled	24	24	17	17	41	20.5
unskilled	31	31	23	23	54	27
Chi square value	7.7312					
Df	3					
P- value	**0.05 (significant)**					

31% of cases were unskilled, 29% skilled, 24% semi-skilled and 16% were employed as clerk. In controls, 32% were employed as clerks, 28% skilled and 23% unskilled workers. This was statistically significant.

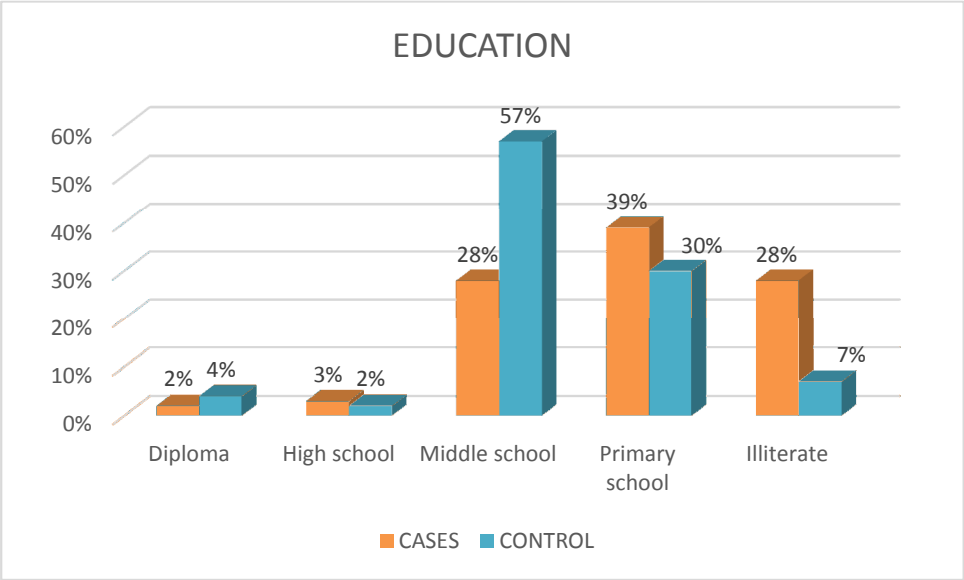

EDUCATION
60%
50%
40%
30%
20%
10%
0%
2%
4%
3%
2%
28%
57%
39%
30%
28%
7%
Diploma
High school
Middle school
Primary school
Illiterate
CASES
CONTROL

TABLE-15: EDUCATION

Education	CASES		CONTROL		TOTAL	
	N	%	N	%	N	%
Diploma	2	2	4	4	6	3
High school	3	3	2	2	5	2.5
Middle school	28	28	57	57	85	42.5
Primary school	39	39	30	30	69	34.5
Illiterate	28	28	7	7	35	17.5
Chi square value	24.5347					
Df	4					
P- value	**0.0001 (significant)**					

In cases the level of education was significantly lower than in controls.

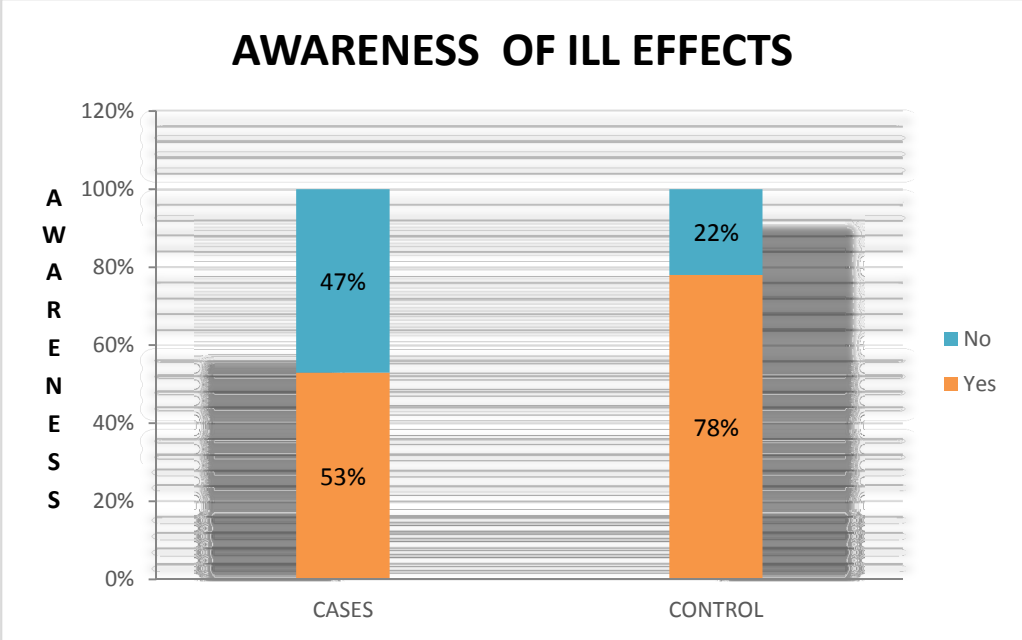

AWARENESS OF ILL EFFECTS
AWARENESS
120%
100%
80%
60%
40%
20%
0%
47%
53%
22%
78%
CASES
CONTROL
No
Yes

TABLE-16: AWARENESS AMONG CASE VS CONTROL

Awareness	Cases (%)	Control (%)	Total	
			N	%
Yes	53	78	83	41.5
No	47	22	117	58.5
ODDS RATIO	2.87			
95% confidence interval for odds ratio	Lower (1.60)	Upper (5.15)		
RISK RATIO	1.86			
95% confidence interval for odds ratio	Lower (1.30)	Upper (2.65)		
CHI SQUARE(Mantel Haenszel test)	13.7598			
P –value (2 tailed)	**0.00 (significant)**			

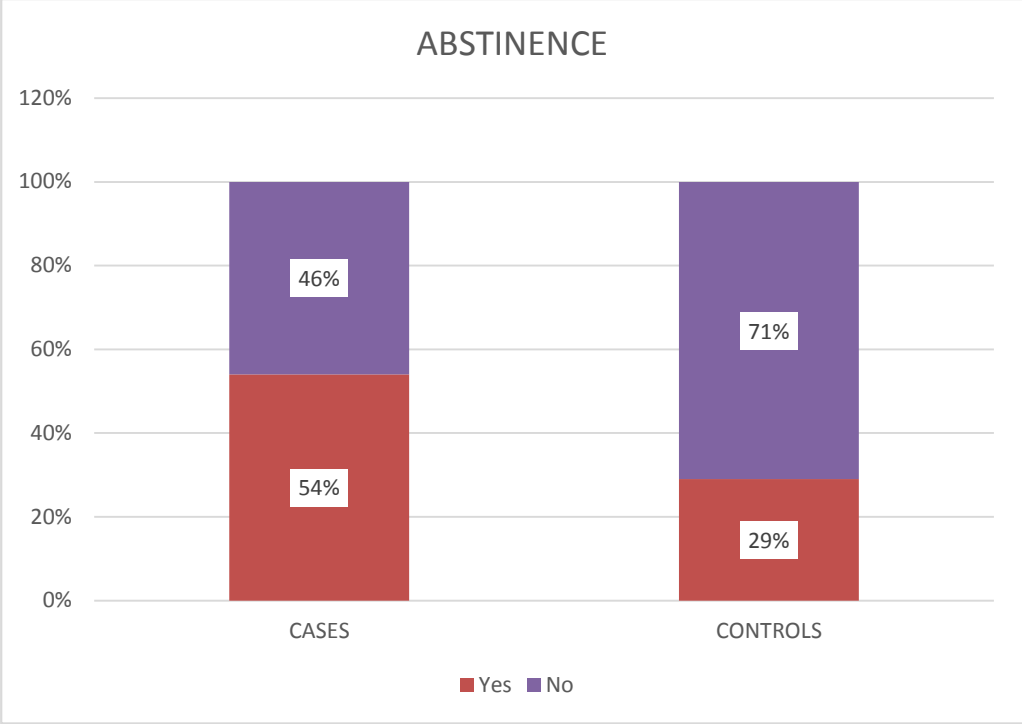

ABSTINENCE
120%
100%
80%
60%
40%
20%
0%
46%
54%
71%
29%
CASES
CONTROLS
Yes
No

TABLE-17: ABSTINENCE IN CASE VS CONTROL

Abstinence	Cases (%)	Control (%)	Total	
			N	%
Yes	54	29	131	65.5
No	46	71	69	34.5
ODDS RATIO	0.318			
95% confidence interval for odds ratio	Lower (0.17)	Upper (0.58)		
RISK RATIO	0.67			
95% confidence interval	Lower (0.54)	Upper (0.83)		
CHI SQUARE(Mantel Haenszel test)	12.80			
P –value (2 tailed)	**0.00 (significant)**			

TABLE-18: ASSOCIATION BETWEEN AWARENESS AND NO.OF QUID USE IN CASE, CONTROL GROUP

Group	Awareness	Number of quid's per day						TOTAL%	Chi-square	P-value
		<5		6-10		>10				
		N	%	N	%	N	%			
cases	Yes	1	1.9	34	64.2	18	34	100	6.36	**0.04 (sig.)**
	No	4	8.5	36	76.6	7	14.9	100		
control	Yes	42	53.8	29	37.2	7	9.0	100	2.27	0.32
	No	14	63.6	8	36.4	0	0	100		

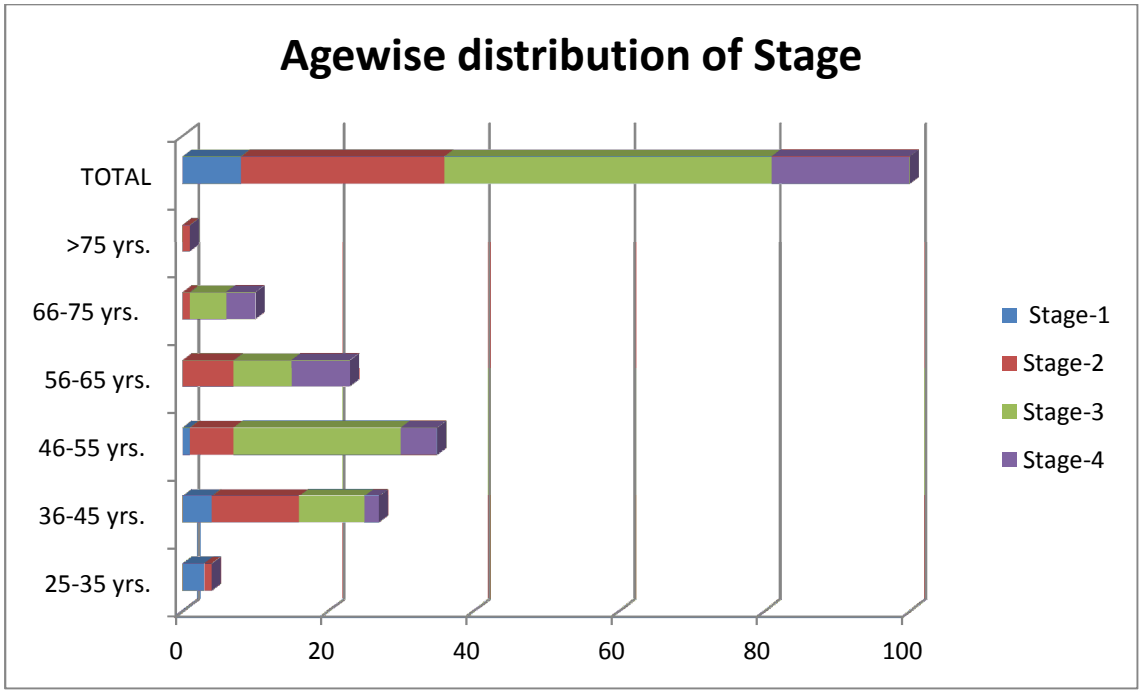

Agewise distribution of Stage
TOTAL
>75 yrs.
66-75 yrs.
56-65 yrs.
46-55 yrs.
36-45 yrs.
25-35 yrs.
0
20
40
60
80
100
Stage-1
Stage-2
Stage-3
Stage-4

TABLE-19: AGE AND STAGING ASSOCIATION

					TOTAL
	Stage-1	**Stage-2**	**Stage-3**	**Stage-4**	
25-35 yrs.	3	1	0	0	4
36-45 yrs.	4	12	9	2	27
46-55 yrs.	1	6	23	5	35
56-65 yrs.	0	7	8	8	23
66-75 yrs.	0	1	5	4	10
>75 yrs.	0	1	0	0	1
TOTAL	8	28	45	19	100
Chi-square	50.6393				
Df	15				
P-value	**0.00 (significant)**				

DISCUSSION

Of the 100 cases in the study, 8% belonged to stage I, 2% belonged to stage II, 45% belonged to Stage III, 19% belonged to stage IV.

The most common age group affected by Squamous cell carcinoma of the oral cavity is between 46-55 years. An alarming thirty one percent of cases presented before the age of 45 years. The mean age of presentation in various studies is between 50 and 55 years (22). In this study where only tobacco quid users were taken into account, the mean age of presentation was 51.15 years.

In regions where tobacco usage is highly prevalent, the mean age of presentation is found to be lower (approaching 35 years). In this study, 31% of cases occurred in people younger than 45 years reflecting the highly prevalent usage in the community.

Previous studies have reported an average of between 20-29 years of exposure to significantly increase the risk of oral cancer which is the combined figure is for all types of tobacco put together. In this study it was found the mean years of exposure in cases was 20.85 years specifically for tobacco quids kept in the mouth.

Studies have reported that usage of more than ten quids per day significantly raises the risk for oral cancer (23). In this study it was found that cases used significantly more number of quids per day than age matched controls.

Nitin Gangane et al in their paper titled “Reassessment of Risk Factors for Oral Cancer” (2), found history of sleeping with quid in mouth to be significantly more in cases. Overnight abuse translates to increased hours of exposure to the tobacco per day. The results of our study is concordant with the above study. It was observed that 39% of cases were regular overnight users of quid compared to only 8% among cases. A total of 69% of cases used to sleep with quid in mouth regularly or occasionally as opposed to 27% of controls who admitted to sleeping with quid in mouth. This finding is statistically significant and further strengthens the dose response relationship between tobacco quid and oral malignancy.

Further strengthening the dose response relationship, is the finding that cases kept each quid for significantly more time in the oral cavity than controls. 38% of cases keep quids for more than one hour in comparison to only 5% of such use in controls. A majority of cases kept the quids for at least more than half an hour (97%) when compared to

controls (51%). A large majority of controls usually reported spitting out the quid within half an hour.(49%)

Multiple substance abuse is significantly more in cases when compared to controls. 52% of cases use bidi occasionally or on a regular basis when compared to controls (20%) and this result is statistically significant (p-0.00). While 80% of the controls have never smoked bidi, only 48% of cases accept to have never smoked bidi.

When it comes to cigarette smoking, 47% of cases are regular or occasional users of cigarette along with tobacco quid as opposed to controls where only 39% have ever used cigarettes. This result was found to be statistically significant (0.03)

53% of cases were regular drinkers of alcohol and 43% of cases were occasional alcohol drinkers (Regular drinkers being defined as people who have more than 3 drinks per week and Occasional drinkers being defined as people who take less than 3 drinks per week) this difference was found to be statistically significant (P-0.00). It seems that regular intake of alcohol along with tobacco quid usage significantly increases the risk of oral malignancy.

History of other smokeless tobacco use like mawa, gutka are significantly more in cases than in controls. A total of 54% of cases confess to occasional or regular use of other smokeless tobacco in the past or in the present when compared to only 29% of controls. 71% of controls claim to have never used other types of smokeless tobacco when compared to the meagre 46% of cases. Thus the usage of two or more types of smokeless tobacco products was significantly more in cases than controls (p-0.00)

There is a significant difference in the occupations of cases and controls (p-0.05). While 55% of cases belonged to the semiskilled or unskilled category, only 40% of controls belonged to this category. 60% of controls belonged to the clerical or skilled category, while only 45% of cases belonged to this category. Thus cases tend to be more from semi-skilled and unskilled group in our study.

When cases and controls were taken together, only a very small percentage of tobacco quid users have seem to have crossed middle school (5% of cases and 6% of controls). Still few were diploma holders (25 in cases and 3% in controls).None of the subjects were graduates.

This shows that most tobacco quid users tend to be school dropouts. When comparing cases and controls, 28% of cases were illiterates as against only 7% among controls. The educational qualification and literacy rate seems to be low in cases than in controls and this has been found to be statistically significant (p-0.0001).

Awareness of the ill effects of tobacco quid seems to be more among cases than controls and there was a statistically significant difference (p-0.00). 78% of controls are aware of the ill effects of tobacco as compared to 53% among cases, the Odds Ratio being 2.87.

There is a high percentage of abstinence among cases than controls. 54% of cases show history of abstinence for the past one year or more when compared to only 29% of controls, the Odds Ratio being 0.318(p-0.00).

Given that the controls are more aware about the ill effects of tobacco quid than cases the abstinence rate should be more among controls. But in this study it is actually the reverse. This can be attributed to the fact that cases tend to get scared once they develop a malignant or premalignant lesion and this fear drives them to abstain. While the

controls who have not experienced any ill effects so far will continue to use tobacco quid.

There was a significant association between age at presentation and stage of the disease. Younger patients (particularly those < 45 years) tend to present with less advanced disease and at an earlier stage when compared to old patients.

LIMITATIONS

- Small sample size precludes us from inferring causality and can merely show associations.
- Cross sectional nature didn't allow us to assess the nature and effect of trend over time.
- We have not quantified the usage of tobacco, hence dose response relationship of these risk factors cannot be demonstrated.
- Our study relies exclusively on the verbal report given by patient with possible chance of recall bias or underreporting.
- The study recruited normal subjects from hospital. It would be more prudent to choose non- cancer relatives of patients who would share common genetic and environmental risk factors, more likely than with general population to minimise the effect of these confounding variables in future studies.
- Female subjects were not recruited and there is high abuse of non-smoking tobacco in females in India.

CONCLUSION

In this study an attempt has been made to study the abuse patterns of oral tobacco quid in patients with oral cancer. Numerous studies have in the past taken into account all types of smoking and non-smoking tobacco products in order to ascertain the role of each type of tobacco in causing oral squamous cell carcinoma with majority concluding that paan tobacco chewing is the most important type of tobacco contributing to malignancy India.

This study specifically concentrated on people who predominantly kept quid in mouth instead of chewing. With the changing pattern of tobacco use, with chewers becoming less and less common , there is a need to reassess the epidemiology of oral cancer with respect to these recent tobacco products.

The study has found that a person who has oral cancer and primarily uses tobacco quids tends to use it more frequently, for longer periods of time than and is more likely to consume alcohol, cigarettes and bidis on a regular basis. These aspects were shown to be statistically significant.

This stresses the synergistic role of alcohol in the disease causation.

With tobacco use becoming more and more prevalent in India with lot of social and medical implications, the need to curb the manufacturing, marketing and use of these products is more than ever.

The disfigurement and loss of function caused by oral cancer, its treatment, the number of life years lost due to the disease and the ever growing incidence of Oral squamous carcinoma in our country point to the fact that we need an effective national policy to identify, treat and rehabilitate these patients. The benefits of early diagnosis and treatment are well known. With new techniques being developed to identify even clinically silent dysplastic changes in the oral mucosa it becomes easier to curb the disease at a very early stage.

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MASTER CHART - CASES

NAME	AGE	SEX	STAGE	YEARS OF EXPOSURE	NO OF QUIDS / DAY	OVERNIGHT USE	AVERAGE TIME PER QUID	BIDI	CIGARETTE	ALCOHOL	OTHER SLT	PROFESSION	EDUCATION	AWARENESS	ABSTINENCE
Rathinam	55	M	3	21	2	2	2	1	2	2	1	6	6	2	1
Kuppusamy	37	M	2	18	2	3	2	1	2	2	1	4	5	1	1
Narayanan	64	M	4	24	3	1	3	2	1	2	2	4	6	2	1
Eswaran	47	M	3	23	2	3	2	1	1	2	2	5	6	1	1
Kannaippan	49	M	3	15	2	2	2	2	3	2	3	6	7	2	2
Dilli Babu	49	M	3	17	2	2	2	1	2	3	1	3	6	1	2
Govindasamy	60	M	4	30	1	1	2	2	1	2	1	3	7	2	2
Vinayagam	47	M	3	22	2	2	2	1	2	3	1	4	6	2	2
Moorthy	42	M	4	22	3	3	2	2	1	3	1	6	6	2	2
Ravi	40	M	3	21	2	3	2	1	2	2	1	4	5	1	1
Munusamy	59	M	4	39	1	1	2	3	1	2	1	5	7	2	2
Kovilpicchai	56	M	2	28	2	1	3	3	1	2	2	5	7	2	2
Anthoni	42	M	3	10	3	3	2	2	1	2	1	4	6	1	1
Venlataramalu	59	M	3	29	1	1	2	3	1	3	2	5	7	2	2
Mangesh	41	M	2	10	2	2	3	1	2	3	1	3	3	1	1
Ellammal	52	F	3	22	2	1	2	1	1	2	1	3	7	2	2
Karmegam	49	M	2	24	2	2	2	3	1	3	1	5	6	2	1
Chakavarthy	56	M	4	26	1	1	2	3	2	2	1	3	6	1	1
Shanmugavel	64	M	3	24	2	1	2	2	1	2	2	3	7	1	1
Thirupathy	42	M	2	10	2	3	2	1	2	3	1	3	5	1	1
Suresh	40	M	3	15	2	2	3	1	2	2	2	4	6	1	1
Ravaniah	50	M	3	25	2	2	2	2	1	2	2	6	7	1	1
boopathy	48	M	3	20	3	2	2	1	2	3	1	3	6	2	1
Thanigachalam	57	M	2	22	2	1	2	2	1	2	2	5	6	1	1
kuppusamy	65	M	4	20	2	1	3	3	1	2	2	5	7	1	1
David	49	M	3	19	2	3	3	1	3	3	2	6	5	2	2
Muivel	53	M	3	18	2	2	2	3	1	3	2	5	6	2	2
parthasarathy	46	M	4	15	3	3	3	1	2	2	1	3	3	1	1
Baskar	49	M	2	12	2	1	2	1	2	3	1	3	5	1	1
Rathinam	70	M	4	30	2	2	2	3	1	2	2	6	7	2	2

Duraikannu	53	M	3	22	2	1	2	3	1	2	2	6	6	1	2
Pencilamma	52	F	3	20	2	1	2	1	1	1	2	6	7	2	2
Venkatesan	47	M	3	12	2	3	3	1	2	3	2	3	5	1	1
Pratap	48	M	2	11	2	2	3	3	2	3	2	4	5	1	1
Sekar	45	M	3	15	2	3	3	1	2	3	2	3	5	1	1
Arunachalam	58	M	3	21	2	3	2	3	1	3	2	6	7	2	2
tirupathi	36	M	2	10	2	3	2	1	2	3	2	4	5	1	1
Gopal	37	M	2	9	3	3	2	1	2	3	1	4	4	1	1
Rana singh	50	M	4	20	2	2	2	2	1	3	2	4	6	2	2
Kumaresan	34	M	2	12	2	3	3	1	2	2	3	5	6	1	2
Bhola	37	M	2	15	2	3	3	1	1	2	2	4	6	2	2
Pakkiri	54	M	4	24	2	1	2	3	1	3	2	6	7	2	2
Muthusamy	58	M	4	29	2	1	2	3	1	3	2	5	7	2	2
Paramasivam	48	M	4	15	2	2	3	3	1	3	2	3	6	2	2
Lourdusamy	66	M	3	30	2	1	3	2	1	2	1	5	7	2	2
Venkatamma	55	F	3	25	2	1	2	1	1	1	3	6	7	2	2
Johnson	48	M	3	26	3	3	3	2	1	3	1	4	6	1	1
Govindaraj	45	M	2	25	2	3	3	2	1	3	2	4	5	1	1
Jagadeesan	62	M	3	30	1	1	3	3	1	3	2	6	7	2	2
Sakthivel	39	M	2	12	2	3	3	1	2	3	1	4	5	1	1
Janagaraj	38	M	1	10	2	3	3	1	2	3	1	4	5	1	1
Kannan	63	M	3	32	2	1	2	3	1	3	3	6	7	2	2
Malathiri	55	M	3	30	3	2	2	3	1	3	2	4	6	1	2
Srinivasan	34	M	1	14	3	3	3	1	2	3	1	5	5	1	1
Datchinamoorthy	51	M	4	26	2	3	2	2	1	3	2	5	5	2	2
Duraikannu	44	M	3	17	2	3	2	3	1	3	2	6	6	2	1
Manikkam	47	M	3	25	2	3	3	1	2	3	2	4	5	1	1
Subramani	66	M	3	30	2	2	3	3	1	2	2	6	7	2	2
Palsami	70	M	4	25	3	1	3	3	1	3	2	3	7	2	2
Siprang	35	M	1	9	2	3	3	1	3	3	1	4	4	1	1
Mohan lal	39	M	1	20	2	3	3	1	1	2	3	4	6	2	2
Senthil	38	M	2	12	2	2	2	1	2	3	1	5	5	1	2
Kamalakannan	60	M	3	26	2	1	2	3	1	3	1	6	6	2	1
Johnson	40	M	3	15	2	3	2	1	2	3	1	3	5	1	1
Murugan	46	M	3	16	3	2	2	1	2	3	2	4	6	2	2
Krishnaveni	57	F	2	23	2	1	1	1	1	1	2	6	7	2	2

Dharani	58	M	2	15	3	1	3	3	1	2	2	5	6	1	1
Anjalai	55	F	2	23	2	1	3	1	1	1	3	6	7	2	2
Pattabiram	39	M	2	14	2	1	3	1	2	3	1	4	5	1	1
Chandrasekar	69	M	2	29	2	2	2	3	2	3	2	6	7	2	1
Subburaj	50	M	3	19	3	3	2	2	1	2	2	5	6	1	1
Alexander	55	M	3	20	2	2	2	1	2	3	1	3	5	1	1
Joseph	40	M	3	14	3	3	2	2	1	3	2	4	6	1	2
Pechiappan	72	M	4	34	2	3	3	3	1	3	2	5	7	2	2
Rajendran	75	M	3	30	2	2	2	3	1	3	1	5	7	2	1
Arulraj	46	M	1	20	3	3	3	1	2	3	2	4	5	1	1
Chellappa	36	M	2	14	3	2	2	1	2	2	1	4	5	1	1
Rajagopal	42	M	4	20	2	3	2	1	2	3	1	4	5	1	1
Kandasamy	60	M	3	28	2	2	2	3	1	2	1	6	6	1	2
Marisamy	55	M	3	16	2	1	3	2	1	3	2	6	7	2	1
Imanullah	45	M	3	17	2	2	2	1	2	1	1	4	6	1	2
Vijayarajan	61	M	4	26	2	2	3	2	1	2	2	6	6	1	2
Ravi	29	M	1	12	3	3	3	1	1	2	2	4	6	1	2
Ratnahar Sahoo	48	M	2	18	2	3	3	1	2	2	1	6	6	2	2
Thulukannam	64	M	4	24	3	2	3	2	1	2	1	6	7	2	2
Iyappan	50	M	3	20	2	3	2	1	2	3	1	5	6	1	2
Surendran	42	M	1	15	3	3	3	3	1	2	2	6	6	1	1
Kulasekaran	56	M	2	22	3	2	2	1	2	3	1	6	6	1	1
Lingasamy	60	M	2	24	2	1	2	2	1	2	1	6	6	2	2
Muthukannan	75	M	2	35	3	2	2	2	1	2	2	6	6	2	1
Jayaraman	72	M	3	37	2	2	2	3	1	2	1	6	6	2	1
Mayalagu	68	M	3	34	2	1	2	2	1	2	1	5	7	1	1
Alagiri	45	M	3	24	3	1	3	1	2	2	1	4	5	1	1
Gurunathan	61	M	3	31	3	1	2	2	1	2	1	6	5	1	1
Saravana babu	43	M	2	17	2	3	3	1	3	2	1	4	5	2	1
Janakiraman	50	M	3	17	2	3	1	1	2	2	3	5	5	2	2
Ezumalai	36	M	1	10	2	3	2	1	1	3	2	5	5	1	1
Venu	47	M	2	13	3	3	1	2	3	3	1	5	4	1	1
Chidambaram	72	M	4	30	2	1	2	2	1	2	2	6	5	2	2
Sivasilam	47	M	2	16	3	2	2	1	1	3	1	5	6	1	1

MASTER CHART – CONTROLS

NAME	AGE	SEX	YEARS OF EXPOSURE	NO OF QUIDS / DAY	OVERNIGHT USE	AVERAGE TIME PER QUID	BIDI	CIGARETTE	ALCOHOL	OTHER SLT	PROFESSION	EDUCATION	AWARENESS	ABSTINENCE
Chinnathambi	52	M	15	1	1	1	1	1	2	1	5	6	1	1
Balachandran	37	M	10	2	1	2	1	1	1	1	4	5	1	2
Jerald Raj	65	M	17	1	1	1	1	1	2	2	3	5	2	1
Kannippan	46	M	12	1	1	2	1	1	2	2	5	6	1	2
Sekar	49	M	10	2	2	1	1	2	2	1	5	7	1	2
Muugan	48	M	9	1	1	1	1	2	2	1	3	5	1	2
Jaganathan	59	M	24	1	1	1	2	1	2	2	3	7	1	2
Pandurangan	49	M	12	2	1	1	1	1	2	1	3	6	2	2
Gnanam	42	M	12	2	3	2	1	1	3	1	4	5	1	2
Vadivel	44	M	11	2	1	1	1	2	2	1	4	5	1	2
Jayabalan	59	M	26	2	1	1	1	1	2	1	6	5	1	2
Bakiaraj	56	M	14	1	1	3	1	1	2	2	5	7	2	2
Dhandayuthapani	44	M	5	2	2	1	2	3	2	1	3	6	1	2
Elango	59	M	20	1	2	1	1	1	2	2	3	5	1	2
Muralimohan	42	M	15	1	2	1	1	1	3	1	3	3	1	1
Janagaraj	52	M	26	1	1	2	1	1	1	1	3	7	1	2
Adhimoolam	48	M	24	2	1	2	1	1	2	1	3	5	2	2
Nandakumar	57	M	20	1	2	1	1	1	2	1	3	6	1	1
Dharman	62	M	14	2	1	2	1	1	2	1	3	7	1	1
Paramasivam	45	M	10	3	1	1	1	2	2	1	3	5	1	2
George	42	M	10	1	1	3	1	2	2	1	3	5	1	2
Shenbagaraj	49	M	25	1	1	1	2	1	2	1	6	5	1	1
Subbu	47	M	11	2	2	2	1	1	3	1	3	6	1	2
Balaraman	57	M	11	2	1	1	1	1	2	2	3	6	1	1
Arunachalam	63	M	10	1	1	1	3	1	3	1	5	5	1	2
Vasanthan	49	M	12	1	1	3	1	1	3	2	4	5	1	2
Vijayakumar	55	M	14	1	1	1	1	1	1	1	5	6	2	2
Sukumar	48	M	11	2	2	1	1	1	2	1	3	3	1	2

Mahohar	54	M	12	1	1	2	1	2	1	1	3	5	1	1
Annasami	73	M	30	2	1	1	1	1	2	1	4	5	1	2
Nataraj	55	M	25	1	1	2	3	3	2	2	3	6	1	2
Kalyanaraman	52	F	10	1	1	2	1	1	1	2	3	5	1	2
Laxmanan	45	M	12	1	3	2	1	1	2	1	3	5	1	1
Arokiya raj	48	M	5	1	1	2	1	2	2	1	4	5	1	1
Ponnusamy	47	M	10	1	1	2	1	2	2	2	3	5	1	2
Saravanan	56	M	12	1	3	2	3	1	3	1	6	5	1	2
Gurunathan	38	M	10	2	1	1	1	2	2	1	3	5	1	2
Krishnakumar	36	M	4	1	1	2	1	1	3	1	4	4	1	1
Danuskodi	49	M	8	2	2	2	1	1	2	2	4	4	1	2
Balaji	34	M	7	2	1	1	1	3	2	1	3	6	1	2
Sambandham	35	M	15	1	1	1	1	1	2	2	4	6	2	2
Rajaram	55	M	20	1	1	2	3	1	3	1	6	5	1	2
Soolai	56	M	20	1	1	2	1	1	2	2	5	5	2	2
Chakrabani	48	M	10	1	2	1	3	1	3	1	3	6	1	2
Kandhan	67	M	30	2	2	2	1	1	2	1	4	5	2	2
Sudagar	55	M	14	2	3	2	1	3	1	1	4	7	1	2
Madhusoodhanan	46	M	16	2	3	1	2	1	3	1	4	5	1	1
Marudhamuthu	45	M	13	3	1	3	1	1	2	1	4	5	1	2
Sekar	62	M	17	3	1	1	3	1	2	2	5	5	1	2
Nagendran	39	M	12	2	3	1	1	1	2	1	4	5	1	1
Palanivel	64	M	5	1	1	1	1	2	2	1	3	5	1	2
Chiokkalingam	63	M	20	1	1	2	3	1	3	1	6	5	1	2
Gnanaraja	52	M	15	3	2	1	1	3	2	2	4	6	1	2
Kumaresan	34	M	10	1	1	1	1	1	2	1	5	5	1	2
Mani	52	M	13	1	3	2	1	1	2	2	5	5	2	2
Anandan	44	M	13	2	3	1	3	3	2	1	6	5	1	1
Santhanam	45	M	12	2	1	3	1	1	2	2	4	5	1	2
Thangappan	66	M	15	1	1	1	1	1	2	2	6	7	2	2
Sitaraman	72	M	19	2	1	1	3	3	3	1	3	5	2	2
Vellappan	35	M	10	2	1	1	1	1	2	1	4	3	1	1

Rasiah	37	M	10	1	2	2	1	1	2	3	4	3	2	2
Appukutty	38	M	12	2	1	1	1	3	2	1	5	5	1	2
Thirumalai	60	M	26	1	1	1	1	1	2	1	6	5	2	1
Mari	42	M	15	2	1	2	1	2	3	1	4	5	1	1
Iyyanar	48	M	16	2	2	1	1	2	2	1	4	6	2	2
Purusothaman	57	M	17	2	1	1	1	1	1	2	6	6	2	2
Thyagarajan	59	M	14	1	1	1	1	2	2	1	6	6	1	1
Somu	52	F	17	1	2	1	1	2	1	1	6	6	1	2
Parattai	36	M	14	2	1	1	1	2	2	1	4	5	1	1
janakiraman	67	M	18	1	1	2	3	2	2	2	6	6	2	2
Parthiban	49	M	10	2	1	2	1	1	2	1	5	6	1	1
Sasikumar	53	M	20	2	2	2	1	2	3	1	3	5	1	1
Palpandi	41	M	10	1	1	2	1	1	3	2	4	6	1	2
Kumaresan	76	M	34	2	1	2	3	1	2	2	5	6	2	2
Ragu	75	M	32	1	2	2	1	1	3	1	5	6	1	2
Manohar	46	M	22	1	1	2	1	2	2	2	4	5	1	1
Viswanathan	35	M	19	1	1	2	1	2	2	1	5	5	1	2
Balaji	44	M	20	2	1	2	1	2	2	1	4	5	1	1
Sonasalam	61	M	13	1	1	2	3	1	2	1	6	6	1	2
Kandasami	54	M	15	1	1	2	2	1	2	2	6	6	1	2
Ramamoorthy	44	M	13	2	1	1	1	1	1	1	4	6	1	2
Ravi	59	M	26	1	1	2	1	1	2	2	3	6	1	2
Thiyagu	30	M	14	1	1	2	1	1	2	2	4	6	1	2
Muniswaran	48	M	10	2	1	2	1	2	2	1	6	5	2	2
Baskar	64	M	14	1	2	2	1	1	2	1	6	5	1	2
Narayanan	54	M	10	2	3	2	1	2	2	1	3	6	1	2
Khonba	40	M	15	3	1	1	1	3	2	1	6	5	1	1
Lingam	57	M	22	2	1	2	1	2	2	1	6	6	1	2
Ritesh	62	M	14	1	1	2	1	1	2	1	6	5	2	2
Sridhar	72	M	20	1	2	1	1	1	2	2	6	5	1	1
Kannan	72	M	21	1	1	2	3	3	2	1	6	6	2	1
Picchiah	66	M	23	1	1	2	1	1	2	1	3	5	1	2
Suresh	42	M	16	3	1	2	1	2	2	1	4	5	1	1
Paneerselvam	61	M	19	1	1	1	2	3	2	1	6	5	1	1
Gemini	41	M	7	1	1	1	1	3	2	1	4	5	2	2

Pratap	52	M	8	1	1	1	1	2	2	1	3	5	1	2
Venkatagiri	39	M	5	1	1	2	1	1	3	2	5	5	1	1
Arumugam	45	M	5	3	1	1	1	3	3	1	5	5	1	2
Divagar	74	M	15	1	1	1	2	1	2	1	6	5	2	2
Ganesan	46	M	10	1	1	1	1	3	3	1	3	5	1	2